STUDY OF CLINICAL, SONOLOGICAL AND HISTOPATHOLOGICAL CORRELATION OF OVARIAN TUMOR

DISSERTATION SUBMITTED FOR

M.D (BRANCH – II)

(OBSTETRICS & GYNAECOLOGY)

APRIL 2013

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that this dissertation titled **"STUDY OF CLINICAL, SONOLOGICAL AND HISTOPATHOLOGICAL CORRELATION OF OVARIAN TUMOR"** submitted by **DR. AISHWARYA JAGAN** to the faculty of Obstetrics and Gynecology, The TamilNadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD degree Branch II Obstetrics and Gynecology, is a bonafide research work carried out by her under our direct supervision and guidance from September 2011 to August 2012.

Dr.UMA, M.D.,D.G.O.,
Professor of Obstetrics and Gynecology,
Department of Obstetrics and Gynecology,
Madurai Medical College,
Madurai.

Dr.P. ANGAYARKANNI M.D.(O.G), DCH,
Professor and Head of the Department,
Department of Obstetrics and Gynecology,
Madurai Medical College,
Madurai.

DECLARATION

I**, Dr. AISHWARYA JAGAN** solemnly declare that the dissertation titled **"STUDY OF CLINICAL, SONOLOGICAL AND HISTOPATHOLOGICAL CORRELATION OF OVARIAN TUMOR"** has been prepared by me. This is submitted to **The Tamilnadu Dr. M.G.R. Medical University, Chennai,** in partial fulfillment of the regulations for the award of MD degree (Branch II) Obstetrics & Gynaecology. I also declare that this bonafide work has not been submitted in part or full by me or any others for any award, degree or diploma to any other university or board either in India or abroad.

.

Place: Madurai **Dr. AISHWARYA JAGAN.**

Date:

ACKNOWLEDGEMENT

I owe my thanks to **The Dean Dr. N. MOHAN M.S.,** Madurai Medical College for allowing me to avail the facilities needed for my dissertation.

I am deeply indebted to **DR. P. ANGAYARKANNI, M.D. (O.G.), D.C.H.,** Professor and Head of the Department of Obstetrics and Gynaecology, Madurai Medical College, Madurai, for her able guidance, inspiration and the encouragement she rendered at every stage of this study.

I am very grateful to my **Former Professor and Head of Department Dr. S. Dilshath M.D., D.G.O.,** and **Prof. Dr. Uma M.D., D.G.O.,** for their valuable guidance in conducting and completing the study. I express my gratitude to other **Professors, Dr. Ambigaimeena, M.D., D.G.O, Dr. S. Geetha, M.D., D.G.O., Dr. T. Uma Devi, M.D., D.G.O. and Dr. Revwathy Kailairajan, M.D., D.G.O,** Department of Obstetrics and Gynaecology for allowing me and helping me in conducting my study in their respective units.

I extend my heartful thanks to **Dr. Usha Ravikumar, M.D.,** Professor and Head of the Department of Pathology, **Dr. N. Sundari M.D.R.D.,** Professor and Head of Department of Radiology and **Dr. Jayaraman D.M.R.D.,** for their guidance throughout my study.

I thank all my **Assistant Professors** for their kind co-operation in helping me to do this study.

Last but not the least I gratefully acknowledge my thanks to all my Colleagues and the co-operation of the patients without whom this study would not have been possible.

CONTENTS

LIST OF CHARTS

LIST OF TABLES

LIST OF PHOTOGRAPHS

INTRODUCTION

Ovary is a very important organ as it is concerned with reproduction.The ovary consists of mesenchymal cells which are multipotential and sex cells which are totipotent.So their stimulation may result in any type of tumor.[1, 2]

Of all gynaecological cancers, ovarian malignancies representsthe greatest clinical challenge because of greater range and variety of tumors with uncertain origin, with no known premalignant lesion and variability in the rate of disease progression.[1] Around 70% of patients with ovarian tumors are diagnosed only at advanced stages due to unavailability of effective screening method and lack of specific clinical presentations at early stage of the disease.

Ovarian cancer is the eighth leading cause of cancer and fifth leading cause of cancer related death in females. Every year 204,000 women are diagnosed to have ovarian cancer and almost 125,000 women die due to ovarian cancer worldwide.[1]

The histiogenesis of ovarian tumor revolves around four main components, mainly surface epithelium, germ cells, sex cord and specialized ovarian stroma.[2]

Ovarian tumors can occur in all ages, but there are differences in the histological types during various decade of life. The predominant type of tumor

during younger age group arethe germ cell tumors. Sex cord stromal tumours occurs in women of all ages.

In premenopausal women 7% of tumors are frankly malignant and 30 % of themin postmenopausal women are malignant.

90 to 95% of malignant tumorsof the ovary are Surface Epithelial Ovarian cancers and the remaining 5 to 10% constitute the Sex Cord Stromal tumors and the Germ cell tumors of the ovary.

Ultrasonography is used extensively to differentiate benign and malignant tumors of ovary.

This study is on the **OVARIAN TUMOR MORPHOLOGICAL INDEXING** – a quantitative analysis relating tumor morphology from sonographically generated images to risk malignancy.

AIM AND OBJECTIVES

1. To analyse the sensitivity and specificity of a morphological scoring system in differentiating benign and malignant tumors of the ovary.
2. To study the epidemiology of ovarian tumors.

REVIEW OF LITERATURE

Ovarian cancer is the second most common of all gynaecological cancers and accounts for 10 – 15 % of gynaecological malignancies in developing countries including India.

Ovarian cancer remains as the leading cause of all cancer related deaths among gynaecological malignancies in United States. The incidence of tumors of ovary is highest in Sweden (19.6/100,000) and the United States (15.4/100,000) and lowest in Japan (10.1/100,000).[3]

Sonography is considered to be the investigation of choice for the evaluation of ovarian tumors due of its high sensitivity, acceptability, and low cost.Campbell et al in 1989 was the first to propose the use of sonography for ovarian cancer screening.[4]He reported a high correlation coefficient of 97% between the ultrasonographically detected ovarian volumes and the actual ovarian volume measured after oophorectomy.

Several studies have been reported to evaluate the benefits of morphologic scoring systems, in quantifying and standardizing the interpretation of sonographic images.

Granberg et al in 1989 concluded that the ultrasound is highly reliable in predicting the characteristics of an ovarian cyst.[5]They reported that the most predictive characteristic feature of malignancy in an ovarian cyst is the papillary projections seen on the inner surface of the cyst wall.

Sassone et al in 1991, suggested an index with four different morphologic features of ovarian cyst architecture, which includes wall structure, septations, cyst wall thickness and echogenicity.[6] He gave a discrete score for each specific character and was evaluated on 143 women with ovarian tumors. The morphological index thus obtained was 100% sensitive and 83% specific in the distinguishing benign tumor from that of a malignant one.

DePriest et al in 1994 recommended a morphologic index system, with only 3 structural characteristics - cyst wall, ovarian volume and septae.[7]This was analysed on 213 women with ovarian tumors. The sensitivity was 89% and specificity was only 70% in predicting ovarian cancer according to his study.

Lerner etalin 1994 used the Sassone classification system and defined the relative importance of the structural components by a multiple regression analysis.[8] Lerneretal appliedan individual score to each of the morphological components by simplifying the indices. The new scoring system was evaluated in 350 women with ovarian masses and reported a sensitivityof 97% and specificity of 77%.

Ferrazzi et al in 1997 designed a new scoring system that was evaluated on 330 women with ovarian masses.[9]According to the study the sensitivity of this scoring system was 87% and the specificity was 67%.

In 2001 Mol et al conducted an external validation of different morphological indices including those by Lerner et al, Granberg et al, Finkler et al, De Priest et al, Sassone et al and Ferrazzi et al.[10]Mol et al conducted a study on 170 ovarian tumor to compare the predictive accuracy of these scoring systems. They reported a lower accuracy than the original scoring indices, with a high sensitivity ranging from 77% to 93% and specificity ranging from 21% to 89 %.

Ueland and collegues in 2003 evaluated the Morphology indexing and concluded it to be an inexpensive and accurate method in differentiating benign ovarian tumors and malignant ovarian tumors, it can be used as an effective tool to plan the management of ovarian tumors.[11] The useof Doppler flow studies along with this morphological indexing have not shown to improve the diagnostic accuracy of morphological indexing.

Persistent ovarian masses and cysts studied in the Hirosaki and Kentucky trials showed a very low likelihood ratio for malignancy tumors - 9.4% in Kentucky trial and 7.0% in Hirosaki trial. By applying the morphological scoring indices to ultrasonographic screening system in the Hirosaki and Kentucky trials,

the reported a high sensitivity ranging from 80% to 90% with a positive predictive value of > 20%.

Since 1980s, the color Doppler flow sonography was considered as an important tool for the predictor of malignancy in ovarian tumors because Doppler flow gives indirect information on the metabolism and direct information on the vascular anatomy of the ovaries.

Hata et al 1999, studied about the colour Doppler assessment of intratumoral blood flow in tumors of the ovary.[12]

According to The National Cancer Data Base Report on Ovarian Cancer,the risks for ovarian cancers were associated with age, nulliparity and a family history of the ovarian tumors. They reported that the women's lifetime risk of developing cancer of ovaryis 1 in 70 (1.4%). Women who have a family history of one or more affected first-degree relatives will have an increase in risk upto 5% - 7%. It is been reported that only 3% -9% of the women with family history will actually manifest hereditary cancer syndromes.

Approximately 90% of malignant ovarian tumors in adult are of epithelial tumors followed by sex cord stromal tumors (6%) and germ cell tumors (3%).

Shruti et al in 2008 studied on the incidence and management of ovarian tumours in 75 cases and reported that 90% of ovarian tumours were of epithelial origin, 75% were benign serous type, 20% of them were mucinous and 2% were endometriod type.[14]

In 2010, GG Swamy et al did a study on 120 cases and concluded that 71.6% were benign, 25% were malignant and 3% were borderline tumors.[15] About two third of all benign neoplasms was seen in patient between 20 to 40 years age, whereas two third of all malignant neoplasms were seen after the age of 40 yrs. The youngest patient in the study conducted by Swamy et al was 12 years of age and the oldest women was of 70 years of age.

HISTORICAL ASPECTS

Herophilus first described the mammalian ovaries, he called it female testis.

Soranus describes the ovary as didymus [twin]. Vesalius and De Fabrica of 1543 labelled ovary as the testis of the uterus. Vesalius, first described the ovarian follicle and corpus luteum.

In 1666, Janswammer dam explained that "the human female testis are comparable to ovaria of birds".

Esmond long in 1761, described all the common tumours of ovary.

Leonardo Da Vinci drew accurately the anatomy of the uterus and ovaries.

Carl von Rokintansky of Vienna, his name is attached to the Rokitansky protuberance of dermoid tumour, also called "dermoid mamilla", "dermoid protuberance" and "embryonic node".

F.Von Werdt proposed the term "granulosa cell tumour" in 1914.

Friedrich Ernst krukenerg described a "fibrosarcoma ovarii mucocellular carcinomatodes" in 1896 under the mistaken impression that the lesion was primary malignancy of ovary. In 1902 Schlagenhauter corrected the error and identified this lesion as metastatic carcinoma.

Struma ovary was first described by Ludwig pick in 1903.

Fritz brenners in 1907 published paper describing 3 cases of Brenner tumour, which now bear his name.

The term “Adenoacanthoma” was coined by Malcolm Dockerty of the Mayo clinic in 1954.

The first histological description as germ cell tumours was given by M. Cherassu in 1906.

Robert mayor first described the mixed and less differentiated forms of tumours which produce viriliszing effect and named as “Adreoblastoma”, in 1930 it was replaced by “Arrhenoblastoma”.

In 1936 Joe Vincetmeigs presented 7 cases of syndrome of ovarian fibroma, ascitie and hydrothorax. In 1937, Rhoads and Terrell suggested the eponym “Meigs syndrome”.

ANATOMY OF OVARY

The ovaries arenodular ovoid structures, located on eachside of the uterus. It is related to the lateral pelvic wall and attached to the posterior layer of the broad ligament, posteroinferior to the fallopian tubes.

They are pink in colour with a smooth surface in young adults. With advancing age they become more greyish with a puckered and scarred surface due to repeated ovulation.They areabout 3 cm long, 1.5 cm wide. The thickness of the ovary is approximately 1cm. The weight of each ovary ranges from 2 - 3.5 gm.[16]

Each ovary has 2 ends – uterine and tubal end. There are two borders – free border and mesoovarium. They have two surfaces – lateral and medial.

RELATIONS:

The upper end is also known as the tubal end. It gives attachment to the fimbrial end of the tubes. It is also attached to the infundibulopelvic ligament. The lower end is also known as the uterine end. It gives attachment to the ovarian ligament.[3.16]

The free border is also called the posterior border. Upper part of free border is related with the uterine tube while it is related to the ureter and internal iliac vessels posteriorly. The mesoovarian border is also called the anterior border. It is

attached to the posterior leaf of the broad ligament. A cleft in this border called the hilum, transmits the ovarian nerves and vessels.

The medial surface of the ovary is related to the fimbrial end of the uterine tube. The lateral surface is convex and lies on the peritoneal depression of the lateral pelvic wall, called the ovarian fossa. The lateral surface is related to the obturator nerves and vessels.

BLOOD SUPPLY:

The ovarian artery is the main arterial supply of the ovary. Ovarian artery arises directly from the abdominal aorta and enters the ovary through the infundibulo pelvic ligament, mesoovarium and mesosalphinx. The ovaries are also supplied by uterine artery.[16,25]

The pampiniform plexus of veins coming out of the hilum form a single vein called the ovarian vein. The left ovarian vein drains into left renal vein and right ovarian vein drains into inferior venacava.

LYMPHATIC DRAINAGE:

They mainly drain into pre and para aortic group of lymph nodes.

NERVE SUPPLY:

Twigs from the aortic plexus pass around the ovarian artery forms the ovarian plexus. Ovarian plexus consists of sympathetic fibres from T_{10}- T_{11} and parasympathetic fibres from the vagus.[16,25]

1. The ovarian surface epithelium (germinal epithelium) forms the outermost layer
2. The cortex is covered by the tunica albuginea, which is lined by germinal epithelium of waldeyer that consist of a layer of cuboidal cells.
3. The ovarian cortex consists of stroma and ovarian follicles.[2,25]
    a. Ovarian follicle : Contains within it the membrana granulosa, cumulus oophorus, the corona radiata, primary oocyte and the zona pellucida. The zona pellucida, antrum, theca of follicle and liquor folliculi are also included within the follicle.
    b. Stromal tissue: The oocytes are included within the stromal tissue, they are derived from the mesenchymal cells and are made up of connective tissue and interstitial cells. They have the ability to respond to hormones like leutinizing hormone or human chorionic gonadotropin.

4. The inner most layer is the medulla. The medulla and cortex cannot be distinctly differentiated. The medulla is made of loose connective tissue derived mostly from mesonephric cells.
5. The hilum (rete ovarii): The hilum is the point where the mesoovarium is attached to the ovary. It contains blood vessels and nerves, and hilus cells which become active in response to steroidogenesis to form tumours.

CELLS OF THE OVARY[3,16] (Fig – 1)

Germ cells: At birth the oocytes represents the germ cells. Germs cellsare capable of reproducing tissue of all germ layers and are considered to be the cells of origin of teratomas.

Granulosa cells: The granulosa cells lie in a single layer around the oocytes. They proliferate under the influence of FSH, forming a fluid that contains the precursor of the zona pellucida.

The Call-Exner bodies,small round masses of dense pink materials surrounded by a rosette of granulosa cells are a specific product of granulosa cells, normal and neoplastic. They synthesize estrogen and various intermediates, including dehydroepiandrosterone. They enlarge at the time of ovulation to form the corpus luteum.

Theca cells: As the maturing graffian follicle enlarge, the immediate surrounding stromal cells enlarge to become rounded and plump, called luteinization. Follicle associated theca cells when activated produce estrogen.

Corpus luteum: The graffian follicle ruptures in response to LH surge and expels the oocyte to become a corpus luteum. The corpus luteum is the main source of progesterone but it also synthesizes estrone and E2, and also androgen.

The corpus luteum producesrelaxin during gestation andpuerperium, probably under the control of HCG.

Hilus cells (HilarLeydig cells): These are clusters of large cells with abundant pink cytoplasm associated with nonmyelinated nerve fibers seen in the hilum of the ovary. They contain crystalloid of Reinke. Their physiological significance of in ovary has not been demonstrated.

Vestigial structure: Persistence of mesonephros as isolated small duct in the mesovarium andaplexiform glandular structure, the rete ovarii are situated on the margin of the ovarian-hilar junction.

ETIOLOGY OF OVARIAN TUMOUR[1,2,3]

The etiology of epithelial cancers of the ovary remains obscure.

1. Geographic factors-more common in women of European and North American origin. Low incidence is seen in Japan.
3. Menstrual factors - early menarche and late menopause
4. Reproductive factors

Nulliparity and low parity are at high risk. There is 40% decrease in risk following first pregnancy, and 14% decrease in risk following subsequent pregnancy.

Elevation in circulating progestin levels during pregnancy protect from ovarian cancer.

5. Oral contraceptives[17]

Long term use of combination of contraceptive reduces the risk of ovarian cancer by 50%. The duration of protection last up to 25 years after the last use.

This is due to the inhibitory effect of the OCP on ovulation. OCP protect against both malignant tumors of the ovaryand functional cysts of the ovarybut not against the benign tumors of the ovary.

6. Dietary factors

There is increased risk with high fats, and low fiber, carotene and vitamin intake.

7. Peritoneal irritants

Increased risk in women working in asbestos related industries and women who used talc on the perineum as they reach ovaries by ascending through the vagina and cervix.

8. Family history

Approximately 90% of inherited cancers of the ovary are due to mutations in the genes BRCA1 or BRCA2.[18]

a. BRCA 1 gene – located on chromosome 17, long arm.Germlinemutation in this gene are responsible for 80 – 90% of hereditary ovarian cancers.

b. BRCA 2 gene –located on chromosome 13, long arm .Germlinemutation in this gene are responsible for 15% of hereditary ovarian cancers.

The lifetime risk of ovarian cancer is approximately 39% in BRCA 1 carriers and 11% in BRCA 2 carriers.

c. Hereditary Site specific ovarian cancer (HSSOC) -this is characterized by early onset <50 years) ovarian cancers especially if there are two or more

first or second degree relatives who have epithelial ovarian cancer. Accounting for 5 – 10 % of all hereditary ovarian tumors.

d. Lynch syndrome II- Hereditary non polyposis colorectal cancer (HNPCC) - this is characterized by early onset (<50 years of age) colon cancer (85%), ovarian cancer (10to12%) and endometrial cancer (40to60%)t. It is caused by mutation in mismatch repair MLH1 and MSH2 and makes up 5 – 10% of all hereditary ovarian tumors.

9. "Incessant ovulation theory"[19]

In nulliparity ovarian surface epithelium form multiple cortical inclusion cysts (CICs) as a result of rupture of follicles due to ovulation that occurs cyclically. The ovarian surface epithelium, undergoes mulllerian metaplasia when they get embedded in the cortex, which on exposure to inflammatory stimuli and hormones results in DNA damage further leading to defined mutation and formation of endometrioid, mucinous and serous tumors of low grade.

10. Other factors

a. Tubal ligation has decreased risk.

b. Increased incidence in women with bloodgroup 'A'.

c. Increased risk in women who had been treated with hormone replacement therapy.

PATHOLOGY OF OVARIAN TUMORS[1,2,3,20.25]

The ovarian tumors are categorized into 3 main types:

1. Surface epithelial-stromal tumours
2. Sex cord-stromal tumours
3. Germ cell tumor

They are classified according to the anatomic structure of origin. Further, each category is divided into a various subtypes. A combinations of these various subtypes found adjacent or mixed together are possibly present with different frequencies (Fig – 2).

WHOHISTOLOGIC CLASSIFICATION OF OVARIAN TUMORS[2.3,20]

I. Surface epithelial-stromal tumors

1. Serous tumors:

a. Benign

   i. Cystadenoma and papillary cystadenoma

   ii. Surface papilloma

   iii. Adenofibroma and cystadenofibroma

b. Borderline malignant (carcinoma of low malignant potential)

   i. Cystadenoma and papillary cystadenoma

   ii. Surface papilloma

   iii. Adenofibroma and cystadenofibroma

c. Malignant

   i. Adenocarcinoma, papillary adenocarcinoma

   ii. Surface papillary carcinoma

   iii. Malignant adenofibroma and cystadenofibroma

2. Mucinous tumors, endocervical-like and intestinal-type:

a. Benign

   i. Cystadenoma and papillary cystadenoma

   ii. Surface papilloma

   iii. Adenofibroma and cystadenofibroma

b. Borderline

   i. Cystadenoma and papillary cystadenoma

   ii. Surface papilloma

   iii. Adenofibroma and cystadenofibroma

c. Malignant
    i. Adenocarcinoma and cystadenocarcinoma
    ii. Malignant adenofibroma and cystadenofibroma

3. Endometrioid tumors:
    a. Benign
        i. Adenoma and cystadenoma
        ii. Adenofibroma and cystadenofibroma
    b. Borderline malignant
        i. Adenoma and cystadenoma
        ii. Adenofibroma and cystadenofibroma
    c. Malignant
        i. Carcinoma
            a. Adenocarcinoma
            b. Adenoacanthoma
            c. Malignant adenofibroma and cystadenofibroma
        ii. Endometroid stromal sarcomas
        iii. Mesodermal (Mullerian) mixed tumours, homologous andHeterologous

4. Clear cell tumors:
    a. Benign: Adenofibrom
    b. Borderline malignant
    c. Malignant: carcinoma and adenocarcinoma

5. Transitional cell tumors:
    a. Brenner tumor
    b. Brenner tumor of borderline malignancy

c. Malignant Brenner tumor
d. Transitional cell carcinoma (non-Brenner type)

6. Squamous cell tumors
7. Mixed epithelial tumors (specify components):
    a. Benign
    b. Borderline
    c. Malignant
8. Undifferentiated carcinoma

II. Sex cord-stromal tumors

1. Granulosa-stromal cell tumors:
    a. Granulosa cell tumors
    b. Thecoma-fibroma group
2. Sertoli-stromal cell tumors, androblastomas:
    a. Well differentiated
        i. Sertoli cell tumour and tubular androblastoma
        ii. Tubular androblastoma with lipid storage and Sertoli cell tumour with lipid storage
        iii. Sertoli-leydig cell tumour
        iv. Leydig cell tumour and hilus cell tumour
    b. Of intermediate differentiation
    c. Poorly differentiated (sarcomatoid)
    d. With heterogenous elements
3. Sex cord tumor with annular tubules

4. Gynandroblastoma
5. Unclassified
6. Steroid (lipid) cell tumors:
    a. Stromal luteoma
    b. Leydig cell tumor
    c. Unclassified

III. Germ cell tumors

a. Dysgerminoma:
    a. Variant-with syncytiotrophoblast cells
b. Yolk sac tumors (endodermal sinus tumors):
    a. Polyvesicular vitelline tumor
    b. Hepatoid
    c. Glandular
c. Embryonal carcinoma
d. Polyembryoma
e. Choriocarcinoma
f. Teratomas:
    a. Immature
    b. Mature
        i. Solid
        ii. Cystic. Dermoid cyst

c. Monodermal and highly specialized
    i. Struma ovarii
    ii. Carcinoid
    iii. Struma ovarii and carcinoid

IV. Gonadoblastoma
    a. Pure
    b. Mixed with dysgerminoma or other form of germ cell tumour

V. Germ cell sex cord-stromal tumor of non gonadoblastoma type

VI. Tumors of rete ovarii

/II. Mesothelial tumors

III. Tumors of uncertain origin and miscellaneous tumors
    a. Small cell carcinoma
    b. Tumour of probable Wolifian origin
    c. Hepatoid carcinoma

IX. Gestational trophoblastic diseases

X. Soft tissue tumors not specific to ovary

XI. Malignant lymphomas, leukemias, and plasmacytomas

(II. Unclassified tumors

III. Secondary (metastatic) tumors

IV. Tumor like lesions

Surface Epithelial Tumors[20,21]

The surface epithelium of the ovary mimics the mesothelium, which forms the inner epitheliallliningof the abdomen and pelvis.

They are classified as

1. Benign - lack of invasive behaviour and exuberant cellular proliferation.
2. Borderline - no invasive behaviour but have exuberant cellular proliferation
3. Malignant- invasive behaviour

Approximately 60% of all tumors of ovary and nearly 90% of malignant tumors of the ovary are Surface epithelial tumors. They usually occur in middle or older women but very rarely can be seen in younger adults, evenbefore puberty.

Surface epithelial stromal tumorshas 5 subtypes:

a. **Serous,**
b. **Mucinous,**
c. **Endometrioid,**
d. **Clear Cell and**
e. **Transitional Cell.**

Malignant epithelial-stromal tumors those who lack specific differentiation are grouped as **Undifferentiated** and those without any specific subtype are classified as **Adenocarcinomas Not Otherwise Specified (NOS)**.

1. SEROUS TUMORS[20,22]

They are formed by the invagination of the surface ovarian epithelium and are lined by tall columnar ciliated epithelial cells resembling those of tubal epithelium. Psammoma bodies characterize serous tumors. They account for nearly 30% of all tumors of the ovary.

a. Benign serous tumors:

They account for 40% of tumors of the ovary. It is bilateral 40% of cases. Occurs in 4^{th} and 5^{th} decade of life (Fig -3).

Gross: They may be cystic, papillary or adenofibromatous on macroscopic appearance. Size may vary from 20 – 30 cms. Typically presents with smooth walled cyst wall with small papillary projections.Papillary serous cystadenoma occurs in combination with a cyst and the papillae on the inner wall or on the outer surface (exophytic). Often associated with ascites and implantation of tumor fragments on the peritoneal surfaces giving a false impression of malignancy.

Microscopy: Columnar epithelium with abundant cilia with microscopic papillae

b. Borderline serous tumors:

They account for 10 % – 15 % of seroustype of ovarian tumors. 30% of tumors are bilateral, 40% of patients have small tumorlets in abdominal and pelvic cavity.

Gross: They have an increased number of papillary projections.

Microscopy: Increased complexity of the papillae of the stromal tissue with epithelial stratification and nuclear atypia.

c. **Malignant serous tumors:**

They account for 75% of epithelial tumors and 25 % of serous tumors are malignant.60% of tumors are bilateral. Usually occurs in 6^{th} decade (Fig -4).

Gross: They have multiple cystic loculations and solid areas. The tumor masses are irregular with solid or papillary areas and capsular nodularity.

Microscopy: Even more complex growth with infiltration of the underlying stroma by solid tumors. Individual tumor cells have extreme degrees of atypia. Histologically, they may be papillary, adenopapillary or of diffuse pattern. Well differentiated carcinoma form glands & have papillary structures. Psammoma bodies are common. Poorly differentiated carcinoma has tumor cells with bizarre

large nuclei called giant cells, the papillae are more complex with a high degree of cellular budding.

2. MUCINOUS TUMORS[20,22]:

Mucinous tumors are made of cells which mimics those of the intestinal epithelium orendocervical epithelium. They account for 20 % – 25 % of all ovarian tumors.

a. Benign

They account for 20 – 25 % of all ovarian tumors. 10% of them are bilateral. Usually occurs in 3rd and 5th decade (Fig -5).

Gross: They are smooth walled, lobulated with whitish or bluish white hue. Cut section shows thick, viscid mucin. The cyst is frequently multiloculated with papillary growth arising from septum.

Microscopy: They are lined by a single layer of tall columnar epithelium with dark staining basal nucleus but without any cilia. The epithelial characteristics are like those of endocervix.

b. Borderline:

They account for 10 %- 15% of all mucinous type of ovarian tumors. These tumors usually occur in 4th and 6th decade of life. Bilaterality is seen in 40% of

endocervical type of borderline tumors where else only 10% of intestinal types are bilateral.

They are similar to benign tumors but may have cystic chambers with papillary areas and few solid areas. Intestinal variety of tumors may rarely be seen in association with pseudo myxoma peritonei.

c. **Malignant**:

They account for 5 – 10 % of all ovarian tumors. Bilaterality is seen in 8 – 10% of tumors. Usually occurs in 6^{th} decade of life.

Gross: They have larger cystic spaces and solid areas with more papillary projections with large areas of hemorrhage and necrosis.

Microscopy: Tumors of well differentiated type are similar to mucin secreting adenocarcinoma of intestinal origin.

3. ENDOMETRIOID TUMOR:[20,21]

They constitute 6 – 8 % of all epithelial tumors. They are formed by cells which are similar to those of the endometrium. They are associated with endometrial hyperplasia, endometriosis or endometrial carcinoma (Fig -7).

a. Benign:

Benign tumors are unusual and they account for only 2% of epithelial tumors. These tumors are mostly unilateral in origin.

Gross: They are large usually measuring 10 – 25 cms. Outer surface is smooth with firm fibrous cut surfaces with variably sized cystic spaces.

Microscopy: These tumors are formed of cudoidal or columnar epithelial cells arranged in a glandular or acinar pattern. They are adenofibromas or cystadenofibromas. The tumor stroma resembles that of ovary rather than that of endometrium.

b. Proliferating:

They exhibit one of following features: multilocular cystadenofibromas or cystic tumours with solid friable nodules.

Microscopy: They are low grade proliferating tumours has glands and cystic spaces embedded in stroma. High grade proliferating tumour shows less stroma, more intra cystic course, papillary and glandular proliferation.

c. Malignant :

Endometrioid adenocarcinoma: Accounts for 15-20% of epithelial ovarian cancer and the second most common histological type. Occurs in 6^{th} decade of life.

13-28% of these tumors have a bilateral origin.

Gross: These tumors have prominent solid areas with cystic spaces. The cystic fluid is bloody or brown.

Microscopy: Well differentiated carcinoma have glandular growth pattern with irregular budding and have good prognosis. Moderately differentiated carcinoma show more complex glandular / micro glandular pattern.Poorly differentiated carcinoma have round, punched out, broader longer papilla with squamous and an adenomatous type of growth pattern.

4. CLEAR CELL TUMORS:[20,21]

Clear cell tumors are uncommon, with most of tumors having malignant potential. They account for 4-5% of all epithelial tumors of the ovary. They are usually seen in the 5^{th} decade of life. Approximately 15 – 20% of tumors are bilateral in origin.

The hall mark of clear cell tumors are large polyhedral cells with eosinophilic granular cytoplasm or peg like ‘hob nail’ cells with little cytoplasm and bulbous nuclei that bulge into lumen of cystic spaces.

a. Benign:

Gross: These tumor areusually cystic with mural nodules. Cystic spaces contain

clear fluid. Average size is 10-12 cm.

Microscopy: Histologically, adenofibromas with tubular spaces lined by one or two layers of hobnail cells, clear cells, or indifferent cuboidal cells. Nuclei are regular in size and shape. Cells contain intracytoplasmic glycogen.

b. Malignant :

Gross: They are large (15-20 cm), thick walled unilocular cysts containing blood stained fluid.

Microscopy: The common type is small to large sheets of polyhedral clear cells separated by delicate fibrovascular septa. Second type is tubulo papillary type. Mitosis are less frequent than in other epithelial carcinoma (2/10 HPF). Eosinophilic hyalinised stroma and hobnail cells are the characteristic features.

5. TRANSITIONAL CELL TUMORS (BRENNERS): [20,21]

Transitional cell tumors are made up of cells that mimicsthe bladder epithelium. They are formed from ovarian surface epithelium that transforms into urothelium like cells.

They accounts for 2% of all primary ovarian neoplasms and are mostly unilateral.

a. Benign

Most brenner tumors are benign. Occur in 5^{th} and 6^{th} decade of life.

Gross: Most of them are very small, well circumscribed firm, solid bosselated tumours with rubbery consistency and faintly lobulated grey white cut surface.

Microscopy: They have sharply demarcated epithelial cellnests set in fibrous stroma (walthard nests), large cystic spaces and large blunt papillae. The epithelial cells are round with clear margins and eosinophilic to clear cytoplasm. The oval nuclei have obvious nucleoli and longitudinal grooving (Coffee bean appearance). Microspaces are characteristic feature of transitional cell carcinomas.

b. Borderline :

Gross: Cut surfaces have variable multicystic component with watery fluid into which friable papillary or polypoid nodules project.

Microscopy: Coarse papillary projections and cysts are lined by multilayered atypical epithelium cells are similar to benign tumours.

c. Malignant :

Gross: They are partly cystic and contain watery or mucinous fluid and have shaggy lining with friable masses projecting from the wall.

Microscopy: These show hetrogenous solid carcinomatous proliferation. Cysts are lined by multilayered epithelium with hyperchromatic and pleomorphic nucleoli.(Fig.-8)

6. **MIXED EPITHELIAL TUMOURS:**

TheWorld Health Organization follows the 10% rule – means that the minor component must form at least 10% of the tumor mass to call it as a mixed tumour.

Common combinations include clear cell carcinoma and endometrioid carcinoma as both these tumours usually originate rise endometriosis.

7. UNDIFFERENTIATED CARCINOMA:

They accounts for 5% of all cancers of the ovary and only 1% of surface epithelial tumors. These tumors areformed by cells with high grade malignant potentials, with high nuclear atypia and loss of cytoplasmic differentiation. Two main types are recognized - the large cell and small cell types. About 50% are bilateral.

II. SEX CORD TUMOURS OF THE OVARY [20,23]

Sex cord-stromal tumors make up 5% of all neoplasms of the ovary. These tumors arise from theca cells, granulose cells, leydig cells and the Sertoli cells.

Sexcord stromal tumors are usually seen in association with certain endocrine manifestations.

A. GRANULOSA CELL TUMOURS

These tumors account for 2 % of all tumors of the ovary. They originate from the rests of primitive granulosa cells unused in folliculogenesis. It is the commonest ovarian stromal tumor. Rokitansky first described these tumors.

The hallmark of these tumours is the presence of cells that resemble those of follicular granulosa or their luteinized variants.

Around 2% of these tumor are bilateral in origin. Inhibin, secreted by granulosa cells to regulate FSH secretion is tumour marker.

There are two majorforms:

- The adult type and
- The juvenile type

a. **Adult granulosa cell tumors**:

Constitutes 95% of GCT, usually occurs in postmenopausal women. Clinical presentations mainly depends on the hormone production, estrogen, they may be associated with endometrial hyperplasia (50%). Unopposed estrogenic stimulation

leads to development of endometrial carcinoma in about 5- 10 % of cases. These tumors are of low grade malignant potential. (Fig.-9)

b. **Juvenile granulosa cell tumors**:

These tumors constitute 5% of all ovarian GCT. These tumors are usually unilateral, and almost 50% of themoccur before puberty. Due to their estrogen production, most tumors result in precocious puberty.

Gross: The external surface is smooth or bosselated. Cut surface appears partly cystic andpartly solid. Solid areas are soft or rubbery in consistency and cystic spaces usually contain some proteinaceous fluid or altered blood.

Microscopy: They exhibitinsular or trabecular, macro follicular or micro follicular patterns. Micro follicular is common - has multiple rounded small spaces made of cystic degeneration of granulosa cells and they contain PAS positive material. These spaces are known as call-exner bodies (30-50%).

B. THECOMA — FIBROMA GROUP OF TUMOURS

These tumors arise from the stromal tissue of the ovary. The thecoma, fibroma and fibrothecoma constitute this group of tumors. These tumors account for 4% of all tumors of the ovaryand they occur in both pre as well as in post menopausal women (Fig -10).

Thecoma:

These tumor cells resemble the ovarian theca interna, mainly composed of spindle cells with lipid elements contained within the cytoplasm. Thecomas are usually seen in postmenopausal age group and are unilateral in origin. Thecomas usually have estrogenic features, suchas postmenopausal bleeding, in some cases may have hyperplasia of theendometrium, and even endometrial cancer. Most of the thecomas are characteristically benign in nature.

Gross: Thecomas vary in size from a nodule to a large, firm rubbery solid tumour. Cut section appears bright yellow to orange in colour. They are almost always unilateral.

Microscopy: They consist of one or two patterns.

- Typical thecomas consists of large ill-defined nodular masses of eosinophilic or vacuolated cells with less fibrous connective tissue. Edema or myxoid change is prominent.

- Second pattern is ‘luteinized thecoma’ — has the appearance of fibroma or typical thecoma with lutein cells.

Fibroma:

Fibromas are the commonest neoplasm of the sex cord tumor group. Although fibroma are said to arise from nonfunctioning stromal tissue with no estrogenic activity, lipid-rich thecoma can have some estrogenic manifestations.

Fibromas usually occur in the middle aged women. Fibromas are mostly benign and few with increase inmitotic activity are may have a malignant potential called the fibrosarcomas.

On rare occasions these tumors may be bilateral and associated with basal cell carcinoma of nevoid type called as the Gorlinsyndrome. They may be seen in association with Meigs syndrome (ascites, ovarian tumor and pleural effusion on right side).

Gross: Fibromas are large tumours with slightly bosselated or smooth serosal surface. They vary in consistency from rubbery to stony hard solid mass. The cut – section appears white and whorled with few regions of cystic degeneration.5% of cases are bilateral in origin.

Microscopy: They consist of variably cellular bundles and intersecting collagenous fibrous tissue.

C. ANDROBLASTOMA (Sertoli — leydig cell tumours) (Arrhenoblastoma) [35]

They account for approximately only 1 % of all sex cord — stromal tumours. They are characterized by presence of sertoli cells, leydig cells or fibroblastic cells. Only <2% in them are bilateral in origin.

1. Sertoli cell tumours

Sertoli cell tumors are formed by proliferation of cells similar to rete ovarii. They are a rare group of sex cord-stromal ovarian tumors constituting<0.5% of all tumors of the ovary.They are usually seen in the 4th decade of life. Sertoli cell tumors are non-function neoplasms, but may occasionally be able to induce sexual developmentprecociouslyby hormone production or rarely may cause virilization in girls.

Gross: Sertoli cell tumors are usually <10cm they are solid, firm, encapsulated typically, yellow or brown, lobulated masses.

Microscopy: They are formed by highly differentiated uniform tubules lined by single layer of cells with basal nuclei and clear cytoplasm. The epithelial cells contain lipid as fine droplets.

d. Leydig cell Tumours (Hilus cell tumour)

Gross: These tumors are mostly unilateral and found in hilar region of ovary and have soft or fleshy consistency.

Microscopy: They are formed of oval or polyhedral cells, which slender rod like bodies with square or tapering ends known as Reinke crystals are present.

e. Sertoli — leydig cell tumours

Sertoli-Leydig cell tumors are made up of cells that mimic stromal and epithelial cells of the testis. They are rare sex cord-stromal ovarian tumors make up<0.5% of all ovarian neoplasms. They usually presents in the 3rd decade of life. These tumors usually cause virilization in about 30% of patients, but they can also rarely produce estrogenic manifestations in few patients.

They are further classified into 5 subtypes:PoorlyDifferentiated, Intermediate Differentiation, Well Differentiated, Mixed and Retiform Type.

Gross: These tumors are usually solid withcompletely or partial cystic areas. The tumors have a yellowish tinge appearance with vesicular or polypoid structures in their inner surface. Most ofthe tumors are unilateral in origin.

Microscopy: Well differentiated tumours have tubular structures lined by cells of sertoli with variable numbers of mature cells of leydig between tubules.

Intermediate differentiation tumours contain cells of sertoli cells, arranged incords, trabeculae or tubules with abundant mesenchymal stroma.

Poorly differentiated tumours are consists sheets of closely packed spindle shaped cells.

D. GYNANDROBLASTOMA:

These tumours show intermingling of well differentiated ovarian and testicular elements. They are usually unilateral in origin.

Gross: Gynandroblastoma are small and solid pole tumours. Cut section shows pink to yellow fleshy nodules.

Microscopy: The ovarian elements resemble mature granulosa cells with call — exner bodies and the testicular element resemble the tubules lined by typical leydig and/ or sertoli cells with Reinke crystals.

E. STEROID (LIPID) CELL TUMOUR

Lipid cell tumors are classified as

- Hilus cell tumor
- Stromal luteomas

- Steroid cell tumors not otherwise specified - NOS

Gross : These appear as a soft, yellowish or yellowish - brown nodules.

Microscopy : The cells are polyhedral or rounded, large mimicking the lutein cells, adrenocortical cells and the leydig cells.

Stromal luteomas:

These are situated in the stroma of ovarian tissue and lack Reinke crystalloids. These tumors are androgenic rarely.

Hilus cell tumours:

These tumors are usually androgenic and presence of Reinke crystalloids is a characteristic feature.

Both Hilus cell tumor and Stromal luteomsa occur usually in postmenopausal age group. These tumors are usually benign.

Steroid cell tumours not otherwise specified:

They are seen in younger age group. Approximately 25 – 40% of these tumors are malignant. They typically produce androgen.

III. GERM CELL TUMOURS[20,24]

Germ cell tumours are the second most common of ovarian tumors only after the surface epithelial tumours of ovary.

GCT arises from totipotent germ cells which are capable of both extra embryonic and embryonic differentiation.

A.DYSGERMINOMA

These are the most common malignant germ cell tumour of the ovary. Peak during 2nd and 3rd decades. Bilateral in 10% of cases. Serum lactate dehydrogenate useful in monitoring individual for disease recurrence.

Gross: Pure dysgerminoma are solid, rapidly growing tumour and with glistening smooth capsule. They may be oval, round or lobulated in appearance. On cut section, solid and varying from firm soft rubbery and from grey pink to yellowish tan colour.

Microscopy: They containaggregates or strands of uniform large cells with lymphocytes, plasma cells, eosinophils, and show a granulomatous reaction with foreign body giant cells and Langhan'scells.

B. YOLK SAC TUMOURS

Yolk sac tumours in its pure form is the most frequent malignant germ cell tumour only next to dysgerminoma. The average age ranges from 16-19 years. Majority are unilateral.Alpha-fetoprotein is commonly produced by these tumours.

Gross: They resemble dysgerminoma with red and yellow areas of necrosis and hemorrhage.

Microscopy: Microscopic features are highly variable. Glomeruloid Schiller — Duval bodies are typical. The presence of diastase-resistant hyaline globules and PAS positive is a characteristic feature of these tumors

C. EMBRYONAL CARCINOMA

Embryonal carcinoma is a raretumour. 60% of the tumours present with hormonal manifestations.It typically produce HCG and 75% also secrete AFP.

Gross: They closely resembles those of yolk sac tumours. Most of the tumours are unilateral.

Microscopy: They resembles testicular embryonal carcinoma.

D.POLYEMBRYOMA

It is rare organoid pattern of embryonal carcinoma and consists of numerous

embryoid bodies, closely resembling early, embryo. Serum AFP or HCG levels or both may be elevated.

E.CHORIOCARCINOMA

Non gestational choriocarcinoma are extremely rare malignant tumours. Choriocarcinomas secrete beta HCG, which are very useful tumour marker.

Gross: Choriocarcinoma are large, rapidly growing tumours and nearly always hemorrhagic.

Microscopy: These tumour are composed of solid aggregates containing a centrally located cytotrophoblast and a peripherally located syncytiotrophoblast.

F.TERATOMA

Benign cystic teratoma:

Ususally occurs in the age group of 20 years to 40 years. In adults they account for 20% of all ovarian tumors and in children they account for 50% of all ovarian tumors.

Usually unilocular with smooth surface. They contain hair and sebaceous material and the wall is usually lined by skin (squamous epithelium). Other

structures commonly seen are teeth, cartilage, bone bronchial mucous membrane and thyroid tissue. Rarely intestinal mucous membrane, pancreas and liver tissue can be seen in the walls of dermoid cyst(Fig – 11).

Immature teratoma (Malignant teratoma):

Among germ cell tumor immature teratoma are the third most common tumour. Usually occurs in children and young adults with a mean age of 18 yrs.

These tumors are usually solid with a trabeculated appearance on cut section. The solid component of the tumor consist of bone and cartilage where else the sebaceous material and hair form the cystic component of the tumor (Fig -12).

Monodermalteratoma

Primitive neuroectodermal tumours: These primitive neuroectodermal tumours (PENTs) are highly malignant neoplasms. Collections of small undifferentiated cells (primitive neuroepithelium) exhibiting varing degree of nuclear pleomorphism characterise the microscopic appearance.

Sebaceous tumours: It's a rare form of a monodermal teratoma. The characteristics necrotic "Cheesy" cyst contents seen in typical teratoma are present but hair is absent.

Cystic struma ovarii: These lesions are seen in women at any age, the average age is 46 years. It is a smooth external surface with small foci of fibrous adhesions. A clear to green down fluid fills the lumen of the cysts.

G.MIXED FORM

The prognosis of these tumors depend on the presence of components of endodermal sinus tumor, teratoma grade III or choriocarcinoma. Tumors accounting for more than one third of these components have a bad prognosis. Tumors with less than one third of these components or when seen in association with teratomagarde I &II, embryonal carcinoma or dysgerminoma have a better prognosis.

IV. GERM CELL -SEX CORD STROMAL TUMOURS

Gonadoblastoma:

These are rare tumour that arise from a dysgenetic gonad. These tumour consist of both sex cord -stromal cells, that have sertoli or granulose cell and immature germ cells.

V. METASTATIC TUMOURS

Metastatic ovarian tumors are very common, accounting for 10% of all malignant ovarian tumours (Fig -13).

Krukenbergs tumour are secondary growths in the ovary. The common sites of primary are - stomach (70%), large intestine (15%) and breast (5%)

Gross :

These tumours are usually bilateral with smooth surface and at times slightly bossed. They are freely mobile, not adherent to the adjacent structures and do not show any capsular infilteration. These tumors maintatin the shape of the ovary. Cut section shows a waxy consistency with occasional areas of cystic changes due to degeneration.

Microscopy:

Stroma appears cellular or myxomatous. With large signetring cells which are ovoid with eccentric nuclei and granular cytoplasm.

Metastatic melanomas and renal cell carcinoma may be confused with granulosa cell tumor and clear cell carcinoma. Leukemias and lymphomas may involve the ovaries usually in their late stage.

VI. OVARIAN TUMOURS ASSOCIATED WITH PREGNANCY[20,26,27]

Benign ovarian tumors are common in pregnancy. The incidence depends on whether the tumor was noted on ultrasound examination – 1 in 50 live births or on pelvic examination – 1 in 80 live birth or those requiring laparotomy – 1 in 1000 to

1500 live births (Fig – 14).

The most common benign ovarian tumor that occurs during pregnancy is benign cystic teratoma (30%) followed by cystadenomas (15%). Malignant ovarian tumors account for only 1 to 2% of all ovarian tumors that complicate pregnancy. The most common malignant ovarian tumor complicating pregnancy is dysgerminoma (60%) followed by epithelial tumors and sex cord stromal tumors.

Whether benign or malignant, most of the ovarian tumors complicating pregnancy are unilateral in origin.

The optimal time for surgical intervention is 16 weeks to 18 weeks. Conservative surgical management for benign tumors has an excellent outcome. The overall 5 year survival rate for malignant ovarian tumors complicating pregnancy depends on the cell type, stage of the disease and on the trimester in which they were diagnosed.

CLINICAL MANIFESTATIONS

Difference between benign and malignant ovarian tumours

		Benign	Malignant
1.	Age	Reproductive age	Extremes of age group (Premenarchal, and post-menopausal)
2.	Rapidity of growth	Slow	Rapid
3.	Pain and tenderness	Absent unless complicated	Present
4.	Surface	Smooth	Irregular
5.	Consistency	Usually cystic	Solid nodular and irregular
6.	Number	Unilateral 15% bilateral	75% bilateral
7.	Fixation	Mobile	Fixty present
8.	Ascites	Usually absent (Except in fibroma)	Present
9.	Oedema of feet	Bilateral	Unilateral with vulvaloedema
10.	Metastasis	Absent	Present
11.	Capsule	Intact	Ruptured
12.	Appearance	Uniform	Variegated
13.	Blood vessels	No engorged vessels on the surface	Large blood vessels present on the surface
14.	Features	Unilocular cyst, few papillary projection, no solid areas	Multilocular, highly papillated solid areas

MATERALS AND METHODS

This is a prospective study conducted in Department of Obstetrics and Gynaecology, in Govt. Rajaji Hospital, Madurai from September 2011 to August 2012.

The study group includes patients who were admitted at Government Rajaji Hospital with an ovarian tumor confirmed by transadbominal ultrasound examination. Totally 136 patients were evaluated during this study period. A standard proforma was used for collection of data.

Transabdominal sonography was performed on all patients using a 3.5 – 5MHztransducer. The ovary was measured in all its three dimensions, and the volume of the ovary was calculated with the use of the ellipsoid formula (length x width x height x 0.523). Cystic ovarian tumor with a papillary projections and solid areas, echogenicity, presence of septum and the presence or absence of free fluid in the extra tumoral space were noted. Morphology indexing was performed according to Ueland and collegues in 2001.

Two different descriptive category were evaluated (Fig15) :

1. Volume of tumor and
2. Morphologic features.

A score from 0-5 was assigned for each of the component. A total score ranging from 0 and 10 for every tumor.

Observations of tumor septa, diffuse echogenicity and extra tumoral free fluid were includedwithin the category of morphological features.

Following morphology indexing,eachtumor was surgically removed and werehistologicallyclassified according to the WHO System of classifying ovarian tumors.

Statistical Tools

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using **Epidemiological Information Package (EPI 2002).**

Using this software, range, frequencies, percentages, means and standard deviations were calculated.

Sensitivity, specificity, accuracy, positive predictive value and negative predictive values were calculated using the following formulae.

$$\text{Sensitivity} = \frac{\text{True positive}}{\text{True positive + False negative}} \times 100$$

$$\text{Specificity} = \frac{\text{True negative}}{\text{False positive + True negative}} \times 100$$

$$\text{Positive predictive value} = \frac{\text{True positive}}{\text{True positive + False positive}} \times 100$$

$$\text{Negative predictive value} = \frac{\text{True negative}}{\text{True negative + False negative}} \times 100$$

$$\text{Accuracy} = \frac{\text{True positive + True negative}}{\text{N}} \times 100$$

RESULTS

A total of 136 ovarian tumors were studied. Table 1 illustrates the incidence benign, borderline and malignant tumors based on histopathological examination. Of the 136 ovarian tumors 92 cases were benign (67.6%), 3 were borderline (2.2%) and 41 were malignant (30.2%).

Table : 1 Incidence of benign , borderline and malignant tumors as per HPE

Type of tumor	Cases	
	No.	**%**
Benign	92	67.6
Borderline	3	2.2
Malignant	41	30.2
Total	136	100

The mean age was 40.5 years (range, 13–85 years).60 patients belong to the age group of 40 years or older and 76patients were less than 40 years of age. The age distribution of the patients are Illustrated in Table 2

Table 2 : Age distribution

Age group (in years)	Number of cases						Total cases	
	Benign		Borderline		Malignant			
	No.	%	No.	%	No.	%	No.	%
11 – 20	6	6.6	-	-	3	7.1	9	6.6
21 – 30	32	35.2	-	-	3	7.1	35	25.7
31 – 40	24	26.4	-	-	8	19	32	23.5
41 – 50	14	15.4	1	33.3	8	19	23	16.9
51 – 60	12	13	2	66.7	14	34.1	28	20.6
Above 60	4	4.4	-	-	5	11.9	9	6.6
Total	92	100	3	100	41	100	136	100
Range	16 – 75		45 – 52		13 – 85		13 – 85	
Mean	36.7		49.7		48.1		40.5	
S.D.	13.1		4.0		15.1		14.6	
'p'	**0.0001** **Significant**							

Table 3 : Parity distribution

Parity	Number of cases						Total cases	
	Benign		Borderline		Malignant			
	No.	%	No.	%	No.	%	No.	%
Pregnant	5	5.4	-	-	-	-	5	5.4
Nulli	20	22	-	-	6	14.3	26	19.1
1	8	8.6	-	-	5	12.1	13	9.5
2	28	30.7	-	-	7	16.7	35	25.7
3	12	13.2	3	100	8	19	23	16.9
4 & above	19	20.8	-	-	15	35.7	34	25
Total	92	100	3	100	41	100	136	100

Table 3 illustrates the parity distribution of ovarian tumours. Ovarian tumors were common in multipara of 2 which is considered as statistically significant. Benign tumors were common in 2^{nd} parity women and malignant tumors were common in women with parity 4 and above constituting 33% and 35.7% respectively. Incidence of benign tumours in nulliparous women was 22% and malignant tumours were 14.3%. There were 5 cases (5.4%) of ovarian tumor complicating pregnancy.

Table 4 : Mode of Presentation

Mode of Presentation	Number of cases						Total cases	
	Benign		Borderline		Malignant			
	No.	%	No.	%	No.	%	No.	%
Mass Abdomen	23	25.3	1	33.3	13	31	37	27.2
Pain	78	84.7	1	33.3	34	82.9	113	83.1
Menstrual Disturbances	3	3.3	2	66.7	5	11.9	10	7.4
Post-Menopausal Bleeding	1	1.1	-	-	-	-	1	0.7
Loss of Weight/Appetite	5	5.5	-	-	4	9.5	9	6.6
Urinary Symptoms	1	1.1	-	-	-	-	1	0.7
White discharge	1	1.1	-	-	-	-	1	0.7
Vomiting	4	4.4	-	-	-	-	4	2.9
Asymptomatic	1	1.1	-	-	-	-	1	0.7
Total	92*	100	3*	100	41*	100	136*	100

*** There was more than one mode of presentation in many cases.**

Table 4 illustrates the mode of presentation of ovarian tumors. The most common presenting features in both benign and malignant ovarian tumours were pain abdomen with an incidence of 84.7% and 82.9% respectively.

Table 5 : Per abdomen consistency of Ovarian Tumor

Consistency	Number of cases						Total cases	
	Benign		Borderline		Malignant			
	No.	%	No.	%	No.	%	No.	%
Cystic	85	93.4	3	100	8	19	96	70.6
Firm	-	-	-	-	5	11.9	5	3.7
Hard	-	-	-	-	17	40.5	17	12.5
Variable	-	-	-	-	10	23.8	10	7.4
Not palpable	7	7.7	-	-	1	2.4	8	5.8
Total	92	100	3	100	41	100	136	100

Table5 illustrates the consistency of ovarian tumor on clinical examination. Most benign tumors were cystic in consistency (93.4%) and malignant tumors were hard in consistency (40.5%) and around 23.8% of malignant tumors have a variable consistency.

Table 6 : Laterality

Laterality	Number of cases						Total cases	
	Benign		Borderline		Malignant			
	No.	%	No.	%	No.	%	No.	%
Unilateral	86	92.3	2	66.7	22	53.6	110	80.9
Bilateral	6	6.6	1	33.3	19	45.2	26	19.1
Total	92	100	3	100	41	100	136	100

Table 6 illustrates the unilateral or bilateral involvement of ovaries in benign and malignant tumors. Most ovarian tumors were unilateral (80.9%). 92.3% of benign tumors were unilateral and 53.6% of malignant tumors were unilateral.

Table 7 illustrates the score for tumors based on the tumor volume by ultrasound.

Score	0	1	2	3	4	5
Tumor volume (cm3)	< 10	10-50	50-100	100-200	200-500	>500

Table 7: USG Volume Score

USG Volume Score	**Number of cases**						**Total cases**	
	Benign		**Borderline**		**Malignant**			
	No.	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
0	1	100	-	-	-	-	1	0.7
1	8	100	-	-	-	-	8	5.9
2	8	66.7	2	16.7	2	16.7	12	8.8
3	23	85.2	-	-	4	14.8	27	19.9
4	27	87.1	-	-	4	12.9	31	22.8
5	25	43.9	1	1.8	31	54.4	57	41.9
Total	92	100	3	100	41	100	136	100
Range	1-5		2-5		0-5		0-5	
Mean	3.54		3.0		4.45		3.84	
S.D.	1.23		1.73		1.11		1.27	
'p'	**0.0001 (Significant)**							

Most benign tumors have a score of $\leq$ 4 and most of the malignant tumors are more have a score of>4

Table8: illustrates the score for tumors based on the tumor structures by ultrasound.

Score	0	1	2	3	4	5
Structure	Smooth wall, sonolucent	Smooth wall, diffuse echogenicity	Wall thickening, <3mm fine septa	Papillary projections, septa> 3mm	Complex, predominantly solid	Complex, solid& cystic areas, extratumoral fluid

Table 8 : Structural Score

Structural Score	**Number of cases**						**Total cases**	
	Benign		**Borderline**		**Malignant**			
	No.	**%**	**No.**	**%**	**No.**	**%**	**No.**	**%**
0	56	96.6	-	-	2	3.4	60	44.1
1	19	90.5	-	-	2	9.5	21	15.4
2	6	75	-	-	2	25	8	5.9
3	5	62.5	-	-	3	37.5	8	5.9
4	2	13.3	1	6.7	12	80	15	11
5	2	8.3	2	8.3	20	83.3	24	17.6
Total	92	100	3	100	41	100	136	100
Range	0-5		4-5		0-5		0-5	
Mean	0.7		4.67		3.88		1.77	
S.D.	1.18		0.58		1.53		2.0	
'p'	**0.0001 (Significant)**							

Most benign tumors have a structural score <2 and most malignant tumors have a structural score > 3

Table 9: Total Morphological Score

Total Morphological Score	Number of cases						Total cases	
	Benign		Borderline		Malignant			
	No.	%	No.	%	No.	%	No.	%
0	1	100	-	-	-	-	1	0.7
1	6	100	-	-	-	-	6	4.4
2	7	100	-	-	-	-	7	5.1
3	19	100	-	-	-	-	19	14
4	19	90.5	-	-	2	9.5	21	15.4
5	20	95.2	-	-	1	4.8	21	15.4
6	12	80	-	-	3	20	15	11
7	3	42.9	2	28.6	2	28.6	7	5.1
8	1	12.5	-	-	7	87.5	8	5.9
9	3	20	1	6.7	11	73.3	15	11
10	1	6.3	-	-	15	93.8	16	11.8
Total	92	100	3	100	41	100	136	100
Range	1- 10		7 – 9		0 – 10		0 – 10	
Mean	4.25		7.9		8.5		5.6	
S.D.	1.9		1.2		2.1		2.7	
'p'	**0.0001 Significant**							

Table 9 illustrates the total of volume and structural score, consolidated as the morphological score.

Most of the benign tumors have a score of < 5 and score of > 5 suggests malignancy.

Table 10: Results as per USG total morphological score and HPE

USG total morphological score	Number of cases	Number of cases					
		Benign		Borderline		Malignant	
		No.	%	No.	%	No.	%
0 – 4 (Benign)	54	52	96.2	-	-	2	3.73
5 – 10 (Malignant)	82	40	48.8	3	3.7	39	47.6

Table 11 : Efficacy of Morphological score with HPE as Gold standard

Result as per Morphological score	Number Of cases	Result as per HPE			
		Positive		Negative	
		No.	%	No.	%
Positive (Score $\geq$ 5)	82	42	51.2	40	48.8
Negative (Score < 5)	54	2	3.73	52	96.2

True Positive	=	42
False Positive	=	40
True Negative	=	51
False negative	=	3
Sensitivity	=	95.5
Specificity	=	56.5
Positive predictive value	=	51.2
Negative predictive value	=	96.3
Accuracy	=	69.1

Table 12 : Morphological Score and Malignancy

USG Morphological Score	No. of cases	Positive cases (Malignant + Borderline) As per HPE result	
		No.	**%**
0	1	-	-
1	6	-	-
2	7	-	-
3	19	-	-
4	21	2	9.5
Less than 5(Negative)	**54**	**2**	**3.7**
5	21	1	4.8
6	15	3	20
7	7	4	57.1
8	8	7	87.5
9	15	12	80
10	16	15	93.8
5 & above (Positive)	**82**	**42**	**51.2**
Total	136	44	32.3

Table 13: Type of Tumor

Type of Tumor	Cases	
	No.	%
1) Epithelial tumors		
a. Serous tumors	74	54.5
- Benign Serous cystadenoma - Benign papillary serous Cystadenofibroma - Borderline serous papillary cystadenoma - Serous cystadenocarcinoma - Papillary Serous cystadenocarcinoma	53 1 2 3 15	39 0.7 1.5 2.2 11.1
b. **Mucinous tumor**	31	22.7
- Benign Mucinous cystadenoma - Borderline mucinous cystadenoma - Mucinous cystadenocarcinoma - Papillary mucinous cystadenocarcinoma	21 1 8 1	15.4 0.7 5.9 0.7
c. **Endometroid tumour** - Benign	2	1.5
d. Brenner tumour - Malignant	1	0.7
e. Undifferentiated tumor	8	5.7
- Poorly differentiated papillary carcinoma - Poorly differentiated carcinoma - Adenocarcinoma -	2 1 5	1.5 0.7 3.5

f. Mixed tumour - Benign papillaryseromucinous cystadenoma	1	0.7
2. Germ cell tumors		
a. Teratoma	11	8.1
1. Immature 2. Mature (Cystic dermoid)	1 10	0.7 7.4
3. Sex cord stromal tumor	6	4.5
a. Granulosa cell tumors malignant **b.** Fibrothecoma	2 4	1.5 3.0
4 Metastatic Carcinoma Krukenbergstumor	1	0.7
5. Soft tissue tumours not specific to the ovary (leiomyosarcoma)	1	0.7
Total	136	100

Table 13 illustrates the incidence of various subtypes of ovarian tumors

In the present study, epithelial tumors (117) were most common ovarian tumors followed by germ cell tumors (11).

DISCUSSION

Ovarian tumours manifest a wide spectrum of clinical, morphological and histological features.Clinically they may be misdiagnosed for other non-neoplastic conditions.

In this study we have analyzed 136 ovarian tumors over a period of one year and correlated their clinical presentation and sonographic finding with the histopathology.

Benign vs. Malignant tumors:

Of the 136 ovarian tumors, according to histologic diagnosis 91(66.9%)of ovariantumors were benign and 42 (30.9%) weremalignant, including 3(2.2%) borderlinetumor masses.

Table 15: Comparison of Incidence of Ovarian Tumors

Study	Benign	Borderline	Malignant
Pilli G et al	75.2%	2.8%	20.74%
Gupta et al	59.4%	0.58%	40%
Couto F et al	80.7%	2.3%	16.9%
Present study	66.9%	2.2%	30.9%

The incidence of benign ovarian tumor (66.9 %) in this study is less than that reported by Pilli G et al and Couto et[29] al but more than that observed by Gupta at al.

Age incidence:

Ovarian cancer may occur at any age. In our study the age incidence was between 13 years and 85 years.

Table 16: comparison of Age distribution of benign tumours [%]

Study	11 -20	21 – 30	31 – 40	41 – 50	51 – 60	>61
Bhatiya et al (1986)	10	38.5	36.6	27.7	15.5	4.4
RamachandranG et al (1988)	9.8	30.7	22	20.4	10.1	4.2
Jagadheswari et al. (1991)	0	16	36	32.3	10.5	5.2
Present study (2012)	6.6	35.2	26.4	15.4	12.1	4.4

In the present study, the peak incidence of benign tumor was between the age group of 21-30 years (35.2 %). Similar observations were made by Bhatiya et al (1986) and Ramachandran G et al(1988) but this incidence is more compared to

that reported by Jagadheswari et al.(1991) in whose study the peak incidence of benign tumors was in the age group of 41 – 50 years.

Borderline tumours were commonly seen in 45- 50 years with a mean age of 49.7 years, as shown in table 2

Table 17: comparisons of Age distribution of malignant tumours [%]

Study	11 -20	21 – 30	31 – 40	41 – 50	51 – 60	>61
Bhatiya et al (1986)	9.8	17.3	27.1	21.8	16.5	3
Ramachandran G et al(1988)	10.5	26.3	29.5	21	9.5	3.2
Jagadheswari et al.(1991)	13.9	16.4	19.9	27.8	17.1	2.5
Present study (2012)	7.1	7.1	19	19	35.7	11.9

In the present study, malignant tumours were commonly seen between the age group 51-60 years with a mean age of 48.1 years. In studies reported by Bhatiya et al and Ramachandran G et al the maximum incidence is seen in 31- 40 years of age, but in Jagadheswari et al the peal incidence is seen in 41-50 years.

Parity:

In the present study ovarian tumours were common in 2nd parity. Of the benign tumours 33% were in 2nd parity.Of the malignant tumours35.7% were in 4th parity and above. Similar observation was found in study done by Shahin Rashid et al and Shah, Vaidya et al in 1990. In the present study we have reported 5 cases of ovarian tumors during pregnancy and all were benign tumors.

Clinical manifestations:

The ovarian tumors manifest with wide variety of clinical manifestation. According to Sharma et al, 93.16% of the cases presented with mass abdomen and 64.9% presented with abdominal pain. In the present study the commonest presenting symptom was pain abdomen (83.1%) in both benign and malignant tumors. 27.2% of patients presented with mass per abdomen. Similar observations were found in the study done by Bhattacharya MM et al[30]&Shahin Rashid et al[31].

Few tumors which produce hormones may cause menstrual disturbances. In our study, 10 cases (7.4%) presented with menstrual disturbances and 1 case (0.7%) with post-menopausal bleeding. According to Pilli et al in 2002, 6.7% of cases presented with menstrual abnormalities which were similar to our study but in contrary to the study conducted by Gupta et al in 1986, in which nearly 40.2% of cases had menstrual disorders as the presenting complaint.

Consistency:

The ovarian tumors vary from cystic to solid in consistency. In the present study 92.3 % of benign tumors were cystic, 40.5 % of malignant tumors were hard and 23.8% of malignant tumors were variable in consistency.In the study conducted by Pilli et al in 2002, of the benign tumors 76% were cystic in the malignant group, 49.2% cases were hard, 44.1% were variable in consistency.

Laterality:

Ovarian tumors may be unilateral or bilateral; bilaterality represents the multicentric origin of the tumor. 6.6% of benign tumors were bilateral and 45.2% of malignant tumors were bilateral. According to Gupta et al 30.2 % of malignant tumors were bilateral.

Histopathological types:

Surface epithelial tumours are common tumours comprising 85.8% of all ovarian tumours. Among epithelial tumors, serous tumors (54.5%) were most common followed by mucinous tumors (22.7%). 53(39%) cases are serous cystadenomas. Histologically they are lined by low cuboidal to columnar epithelium.1 cases of cystadenofibroma was present. It was cystic and lined by low cuboidal epithelium.

Only 2 cases (1.5%) of borderline serous tumours were reported. One was unilateral and the other was bilateral, both were cystic in consistency. Histologically it has stratification of epithelial cells with mitotic activity and stromal penetration.

19 malignant serous tumours were present. 10 were unilateral and 9 were bilateral. On gross examination 6 were cystic, 8 were solid and 4 were variable in consistency. Microscopically they are lined by more than one layer of columnar epithelium with nuclear polymorphism and hyperchromatic nuclei.

21 mucinous cystadenoma were present, out of which 16 were multilocular and 5 were unilocular. The content was mucoid. The epithelial lining of the cyst wall is columnar with basophilic cytoplasm and basal nuclei. There was 1 case of borderline mucinous tumour, 8 cases of mucinous cystadenocarcinoma and 1 case of mucinous cystadenocarcinoma with papillary differentiation (Fig 16).

Germ cell tumours

These are second most common group of tumours. In germ cell tumors, mature benign cystic teratomas (Dermoid cyst) (7.4%) were most common.

The common age group was 20-30years. 2 patients were in their 5^{th} decade of life. Grossly they were cystic. Majority of tumours have stratified squamous epithelium and dermal appendages like hair, sebaceous glands etc.

Sex cord stromal tumours:

2 case of granulosa cell tumour presented with menstrual disturbances and both were malignant and solid in consistency.Histologically the cells appear polygonal to round with hyperchromatic nuclei with central groove and a very scanty cytoplasm.

4 cases of fibrothecoma presented with mass abdomen and menstrual irregularities.Grossly it was variable in consistency and unilateral.Histologically it consists of lipid rich cells resembling theca cells.

Mixed tumours:

Only 1 case of benign seromucinous cystadenoma with papillary differentiation was reported.

Metastatic tumor:

There was one case of krukenbergs tumor; the patient was operated for carcinoma stomach 4 years back following which she presented with mass abdomen.

Soft tissue tumours not specific to the ovary:

1 case of leiomyosarcoma has been reported in a 40 year old P3L3 presented with a mass abdomen. On examination, it was a irregular mass of size 20x20 cm variable consistency and restricted mobility with no ascites, per operatively Left ovary replaced by a fleshy vascular tumour of size 20x18 cm multiloculated with variable consistency. On histopathology the Cells were spindle shaped cells with elongated nuclei and eosinophilic cytoplasam of interlacing bundles with whorled appearance. Pleomorphism, hyperchromatic nuclei with of mitoses 14/10 hp were seen(Fig 17).

Ultrasound examination:

With the demand for Evidence Based Medicine it has become a challenging task to develop a morphological index to predict the malignancy of ovarian mass without any surgical intervention. An ideal scoring system must be accurate, easy to interpret, time saving, less interobserver bias and cost effective. The use of such a scoring system must help in triaging the women with malignancy to an oncologists and avoid unwanted surgical intervention in women with benign tumors.

Several investigators have put forth many scoring system to compare the morphological features of the tumors. The present Morphological Indexing is a modification ofthescoring system proposed by DePriest and colleagues[32].

Morphological scoring system:

In the present study, one hundred and thirty six patients with ovarian tumor were evaluated sonographically and were operated during the study period from September 2011 and august 2012. The morphological characters were evaluated in two different descriptive characters:

1. Volume of the tumor volume and
2. Morphological features.(Fig.18a-g)

A score from 0-5 was assigned for each of the component.A total score ranging from 0 and 10 for every tumor.

According to the present study the benign tumors have a mean volume score of 3.58 which is statistically significant (table 6). This mean deviation to the higher value is mainly contributed by the benign mucinous cystadenoma which were larger in size with a volume of > 500. The malignant ovarian tumors have a mean volume score of 4.45 which is statistically significant. Of the 42 malignant tumors 31 were of volume > 5, only 2 tumors had a score 2 which were both serous cystadenocarcinoma.

In the present study, benign tumours had a mean structural score of 0.7 and malignant tumors had a mean structural score of 3.88 (table 7). Of the benign tumors most of them had a smooth wall with either sonolucent or diffuse echogenicity accounting for a score of 0 or 1. Of the malignant tumors most of them had a complex, solid and cystic areas with extramural fluid accounting for a score of 5.

The total morphological score was calculated by the sum of volume and structural score and tabulated in table 8. The benign tumors had a mean score of 4.25 and malignant tumors had a mean of 8.5 which was statistically significant with a p value = 0.0001). The malignancy risk is directly related to the volume of the tumor and the various structural components each with a p value = 0.0001.

There were only 2 cases of malignancy (3.7%) reported with a tumor morphological score of< 5 of the 54 ovarian tumors with a morphological score <5 studied , compared to only 0.3% as stated in Ueland et al in 2003. In contrast there were 42 cases (51.2%) of malignancy with a tumor morphological Score $\geq$ 5 of the 82 tumors studied, compared to only 41% as stated by Ueland et al in 2003.

In the present study the morphological index score of≥ 5 as a predictor of malignancy has statistical parameters as follows:

- Sensitivity = 95.5
- Specificity = 56.5
- Positive predictive value = 51.2
- Negative predictive value = 96.3
- Accuracy = 69.1

These parameters were much less than that observed by Ueland et al in 2003 who reported a sensitivity of 98.1%, specificity of 80.7% , positive predictive value of 40.9%, negative predictive value of 99.7%, and accuracy of 82.8%.

Table:18 Comparison of Sensitivity and Specificity with other Studies

Source, Year	Sensitivity	Specificity
Carter et al,1988	0.62	0.95
Granberg et al, 1991	0.82	0.92
Sassone et al, 1991	1.0	0.83
Lerner et al, 1994	0.97	0.77
DePriest et al, 1994	0.89	0.70
Yamashita et al, 1997	0.85	0.78
Ferrazzi et al,1997	0.75	0.67
Mol et al, 2001	0.77 – 0.93	0.21 – 0.89
Present study	0.93	0.56

Table 18 illustrates the sensitivity and specificity of various studies. These individual studies to observer bias. Mol et al[10] in 2001 did an external validation of these sonological indices and revealed a lower accuracy with a high sensitivity ranging from 77 - 93% only at the expense of a wide range in specificity ranging from 21 - 89%. In the present study the sensitivity is 93% and specificity is 56%

SUMMARY

A total of 136 ovarian tumors were analysed and of these 136 ovarian tumors91 cases were benign (66.9%), 3 were borderline (2.2%) and 42 were malignant (30.9%) based on histopathology.

The mean age was 40.5 years (range, 13–85 years).60 patients belong to the age group of 40 years or older and 76 patients were less than 40 years of age.

Ovarian tumors were common in multipara of 2 which is considered as statistically significant. Benign tumors were common 2^{nd}parity and malignant tumors were common in 4^{th} parity and above constituting 33% and 35.7% respectively.

Incidence of benign tumours in nulliparous women was 22% and malignant tumours were 14.3%. There were 5 cases (5.4%) of ovarian tumor complicating pregnancy.

The most common presenting features in both benign and malignant ovarian tumours were pain abdomen with an incidence of 84.6% and 83.3% respectively.

Most benign tumors were cystic in consistency (93.4%) and malignant tumors were hard in consistency (40.5%).Around 23.8% of malignant tumors have a variable consistency.

Most ovarian tumors were unilateral (80.9%). 92.3% of benign tumors were unilateral and 53.6% of malignant tumors were unilateral.

In the present study, 117 cases (85.8%) were epithelial tumors and were the most common ovarian tumors followed by germ cell tumors, being reported in only11cases (8.1%).

The morphological scoring system as assigned by Ueland et al was used to calculate the morphological index.

A particular score is assigned to each depending on the tumor volume. Most benign tumors have a score of ≤ 4 and most of the malignant tumors have a score >4. A particular score is assigned to each depending on the tumor structure. Most benign tumors have a structural score of <2 and most malignant tumors have a structural score of > 3.The morphological index for Most of the benign tumors was< 5 and score of ≥ 5 suggests malignancy.

The present study has the following statistical parameters:

True Positive	=	42
False Positive	=	40
True Negative	=	51
False negative	=	3
Sensitivity	=	95.5
Specificity	=	56.5
Positive predictive value	=	51.2
Negative predictive value	=	96.3
Accuracy	=	69.1

CONCLUSIONS

The findings of the present studyconclude the following

- Morphological scoring system is helpful in identifying women with ovarian tumors who are at risk for malignancy.
- Morphological scoring system also effectively decreases the need for unwanted surgery in benign ovarian tumor.
- It is easy to perform.
- This scoring system is subjected to interobserver variation.
- Morphologic indexing helps to standardise the ultrasound reading without adding costs.

Chart 1

Incidence of benign , borderline and malignant tumors as per HPE

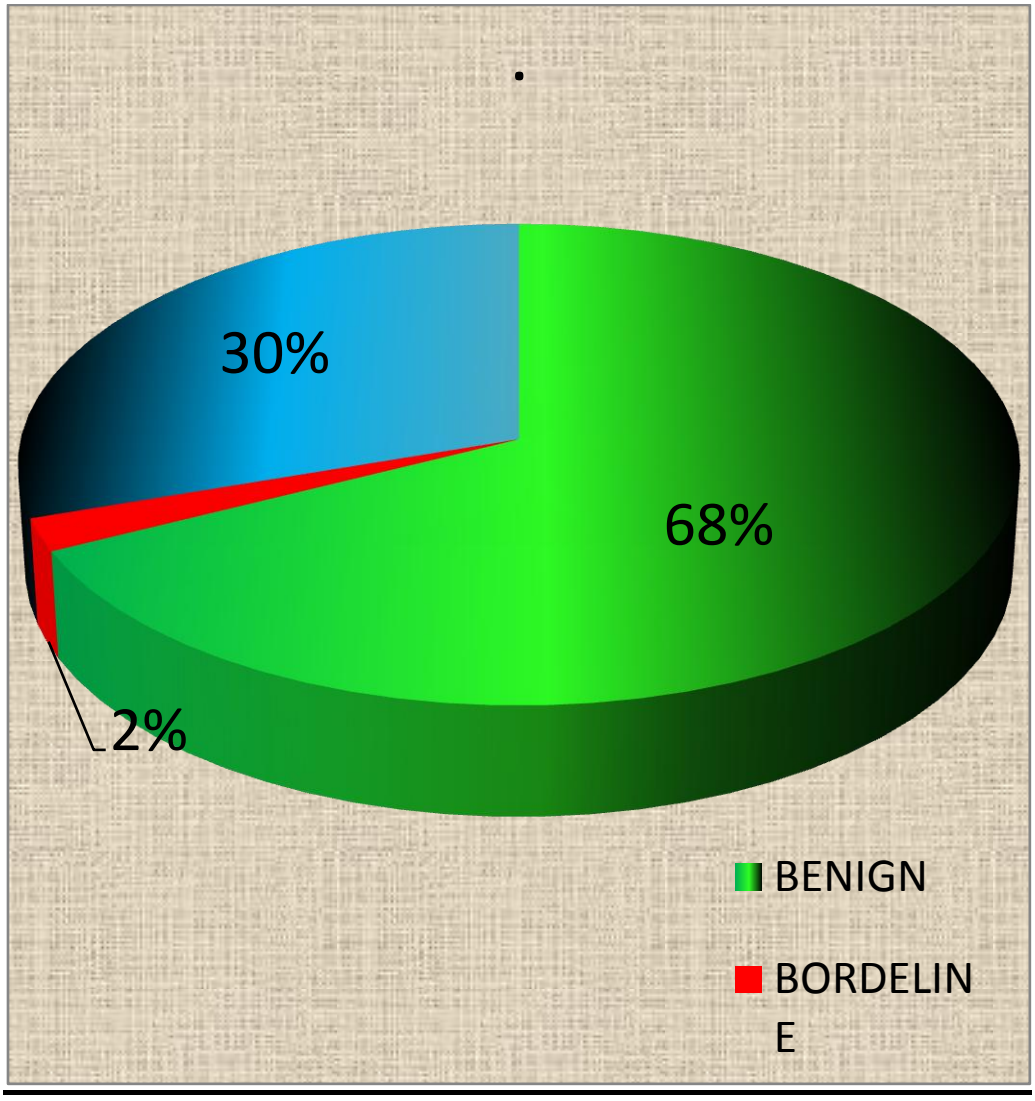


Chart 2 : Mean Age Distribution

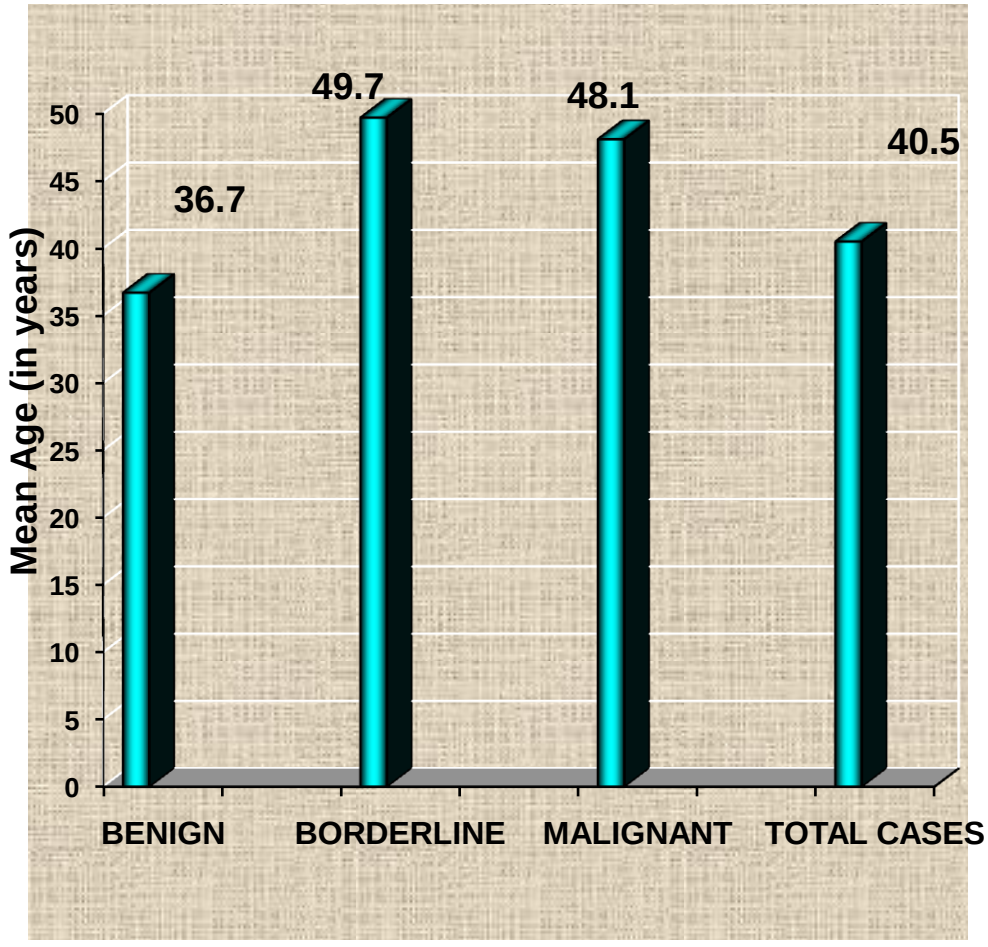


Chart 3: Parity distribution

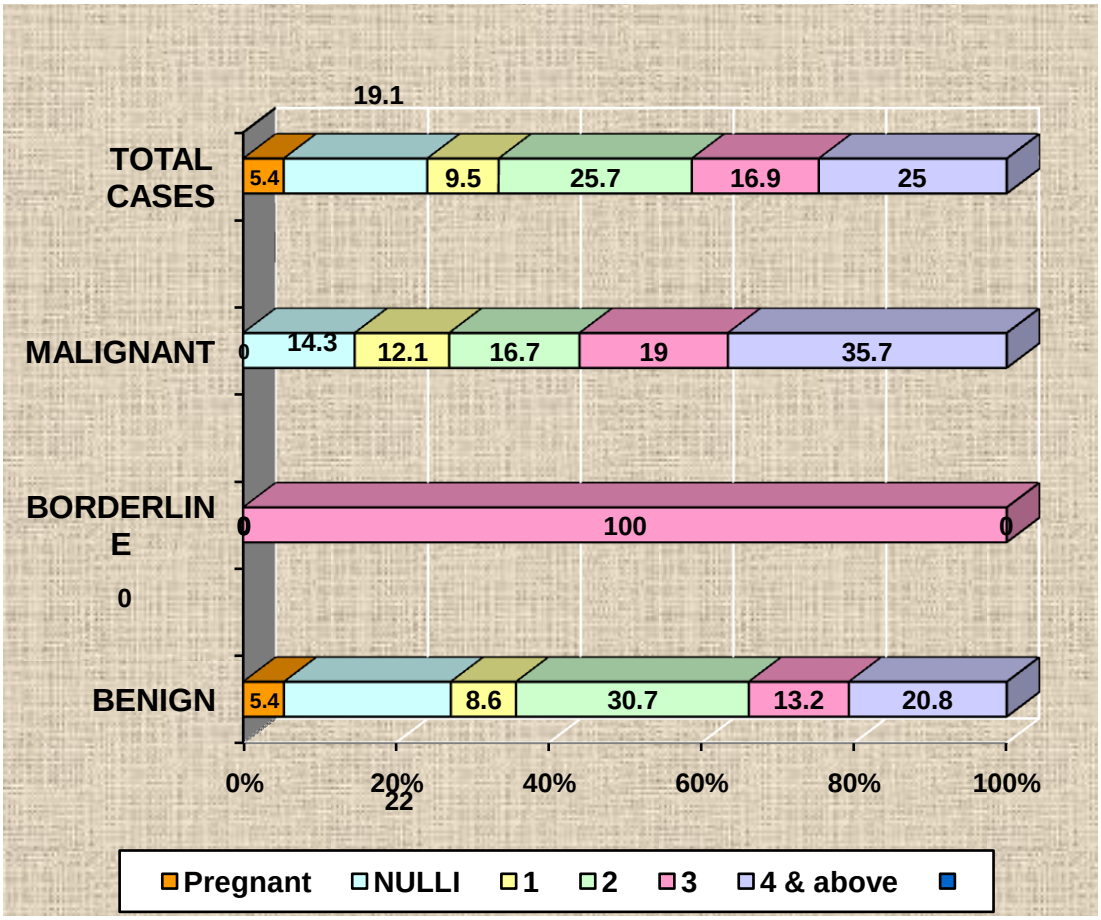


Chart 4: Per abdomen c

onsistency of Ovarian Tumor

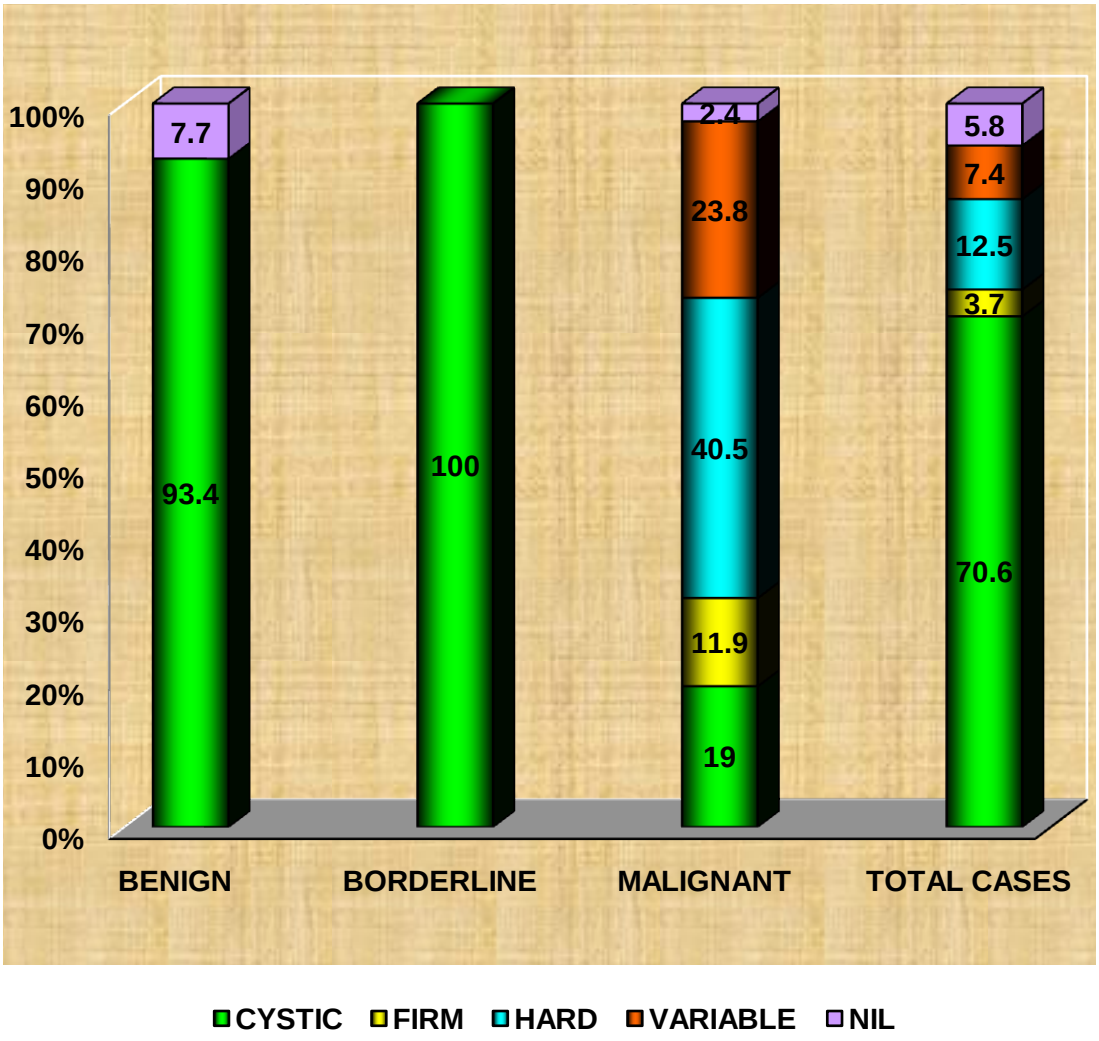


Chart 5: Laterality

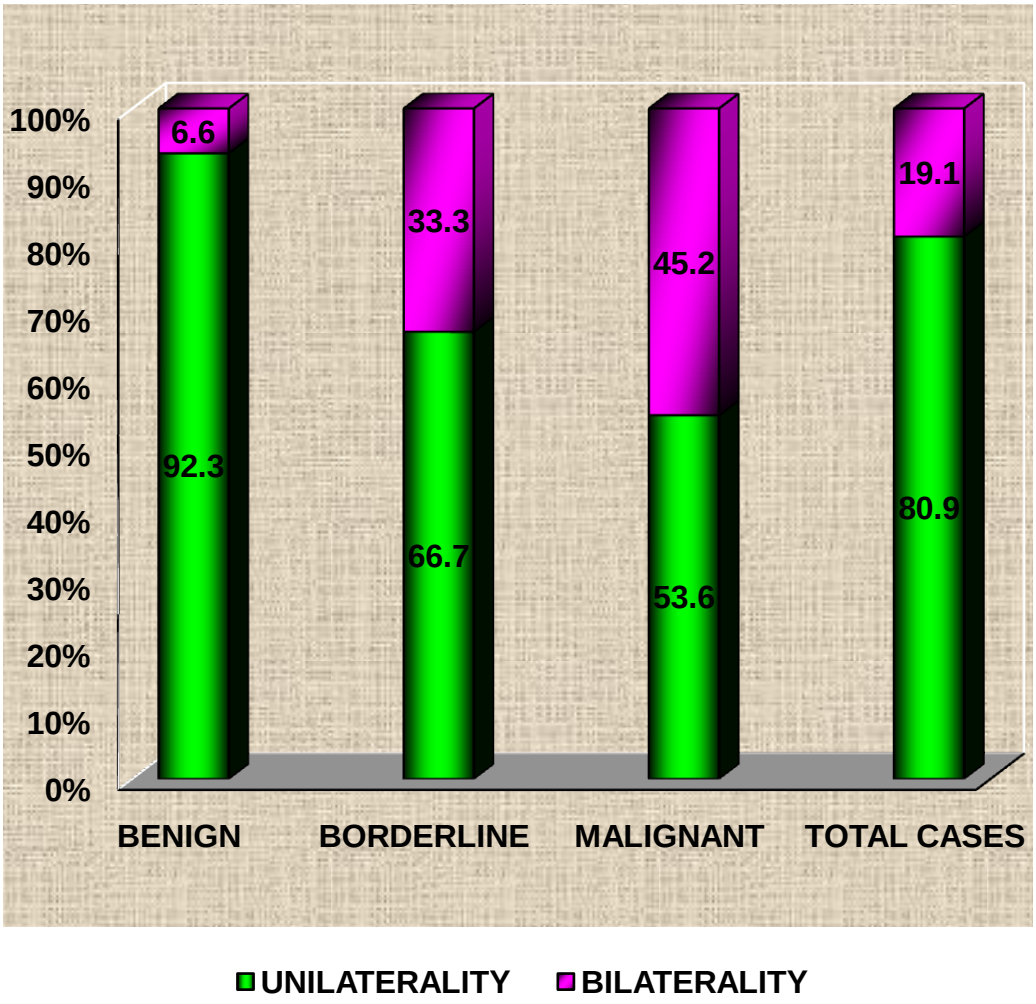


Chart 6: USG Volume Score

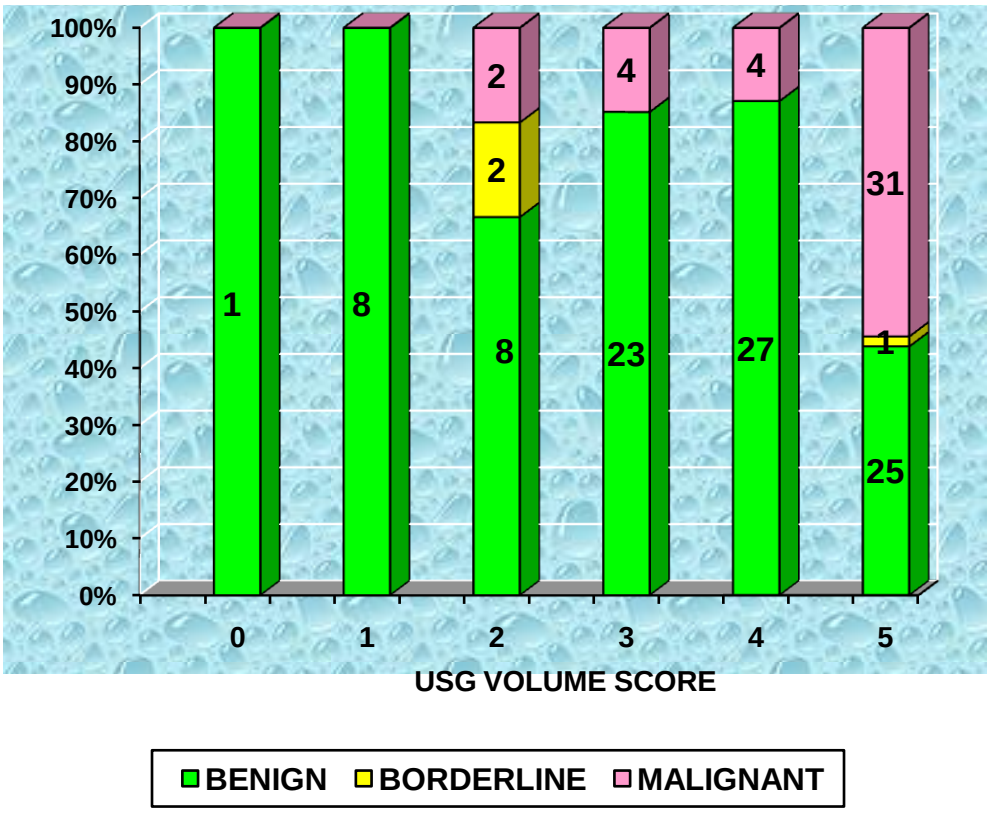


Chart 7: Structural Score

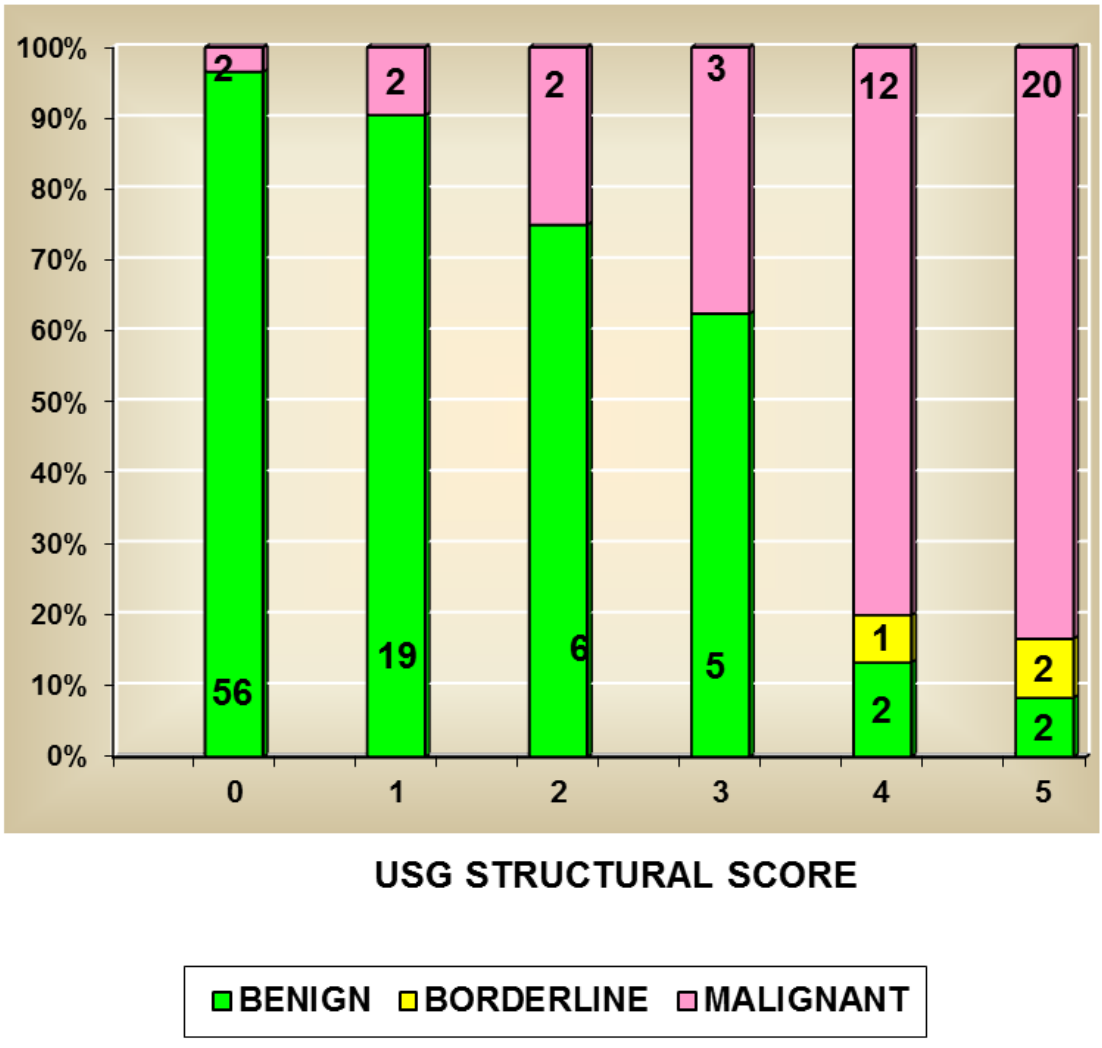


Chart 8:

Morphological score

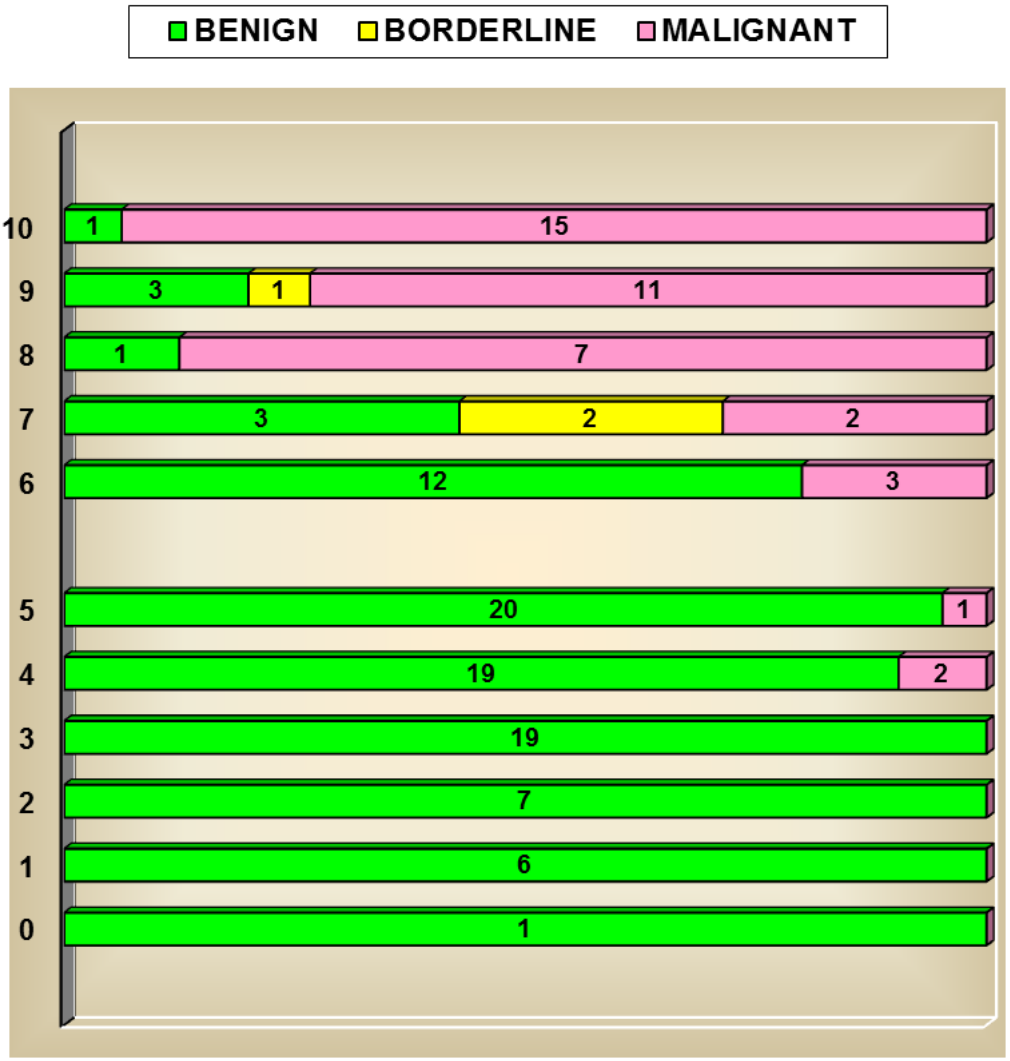


Chart 9:

Results as per USG total morphological score and HPE

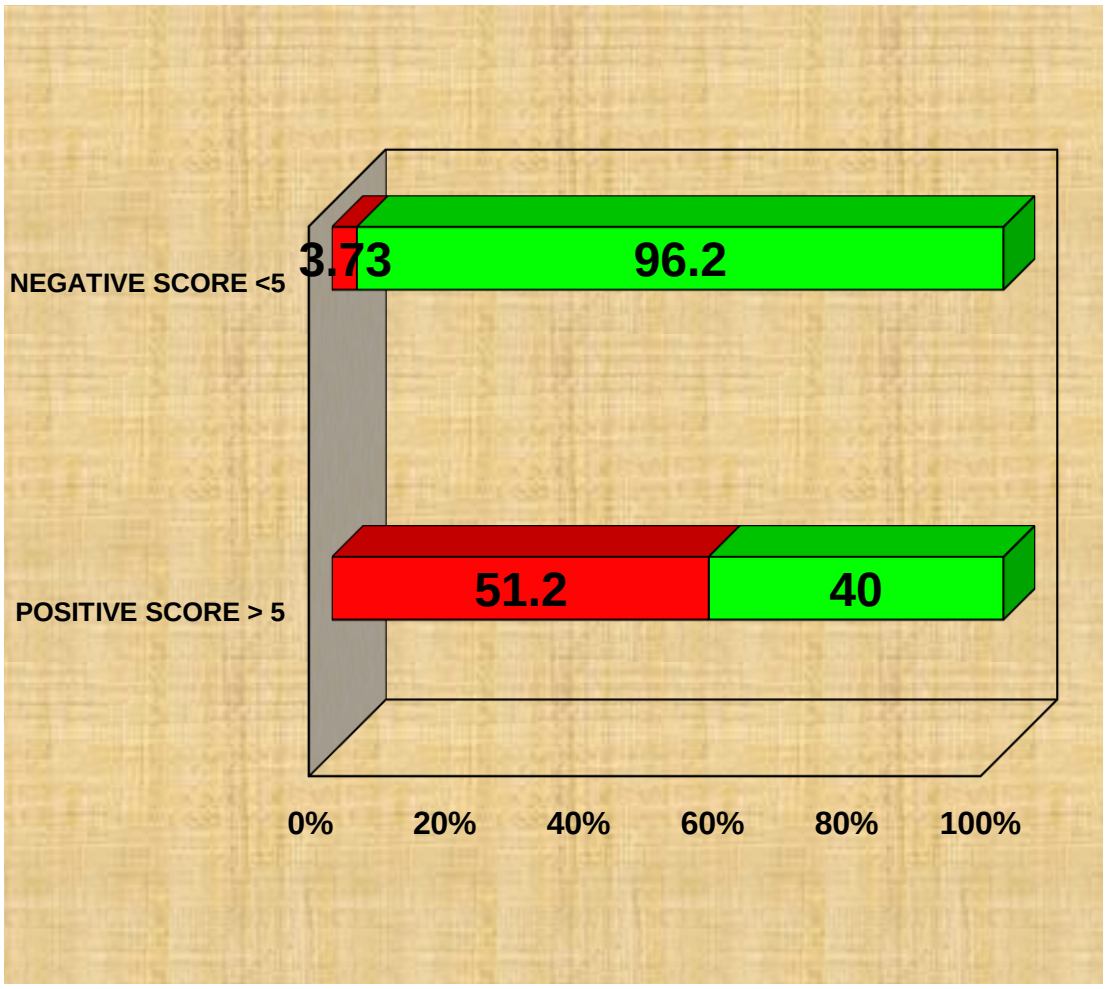


Chart 10:

Comparison of Sensitivity and Specificity with other Studies

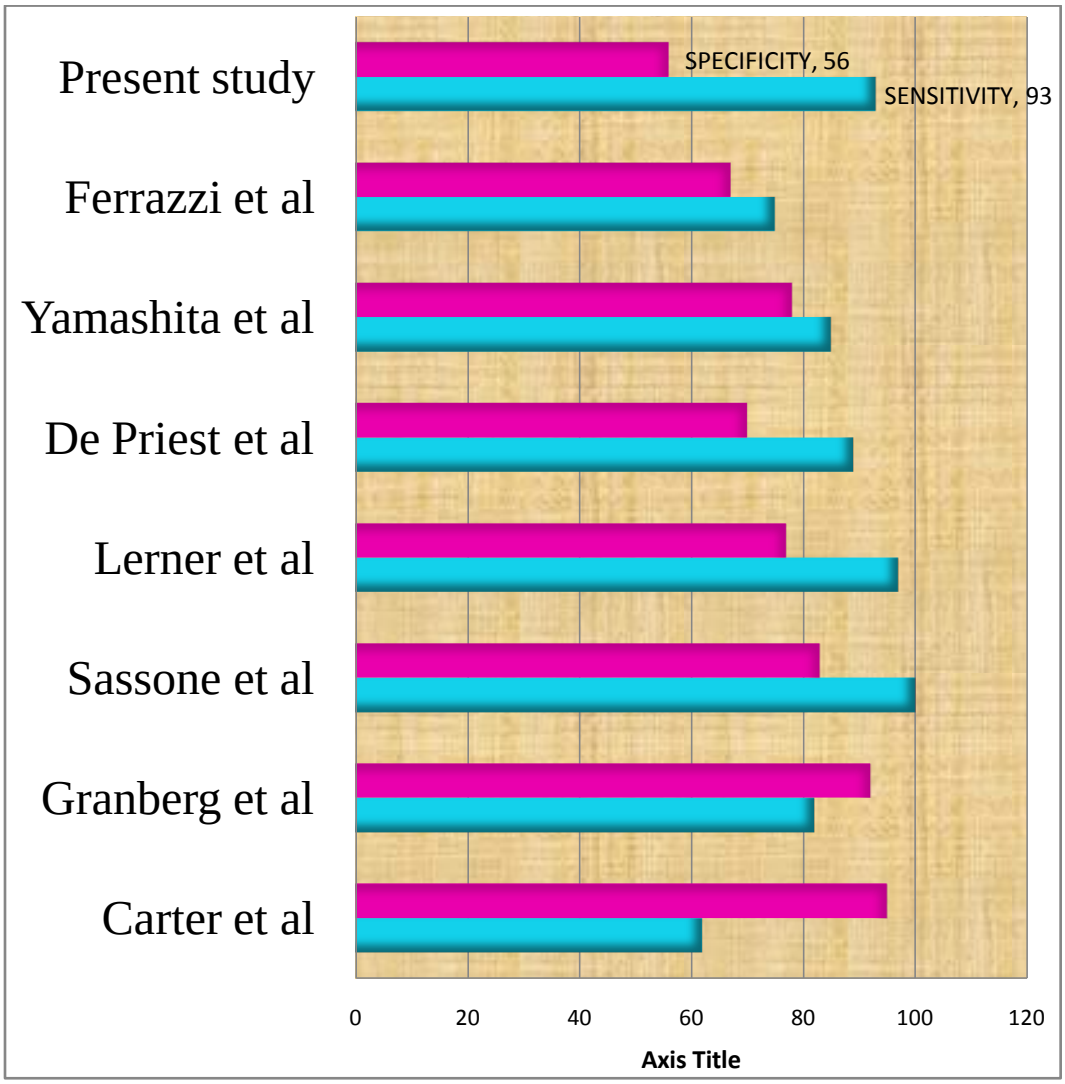
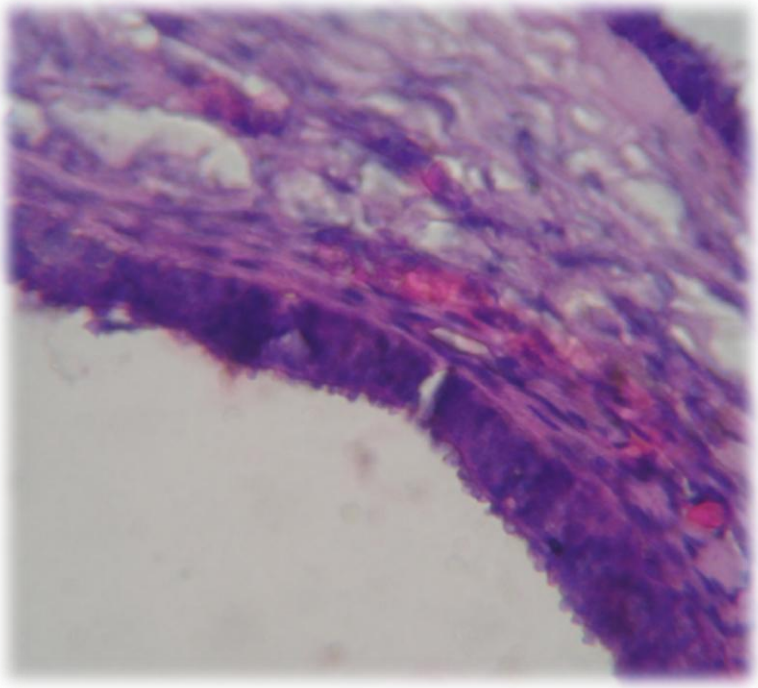
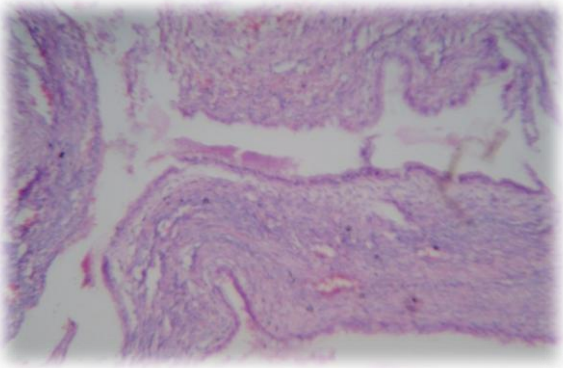


Fig 3.SEROUS CYST ADENOMA

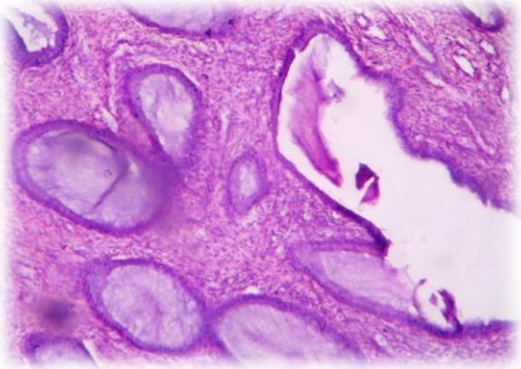


Stromal papillae and columnar epithelium with abundant cilia

Fig 5.MUCINOUS CYSTADENOMA

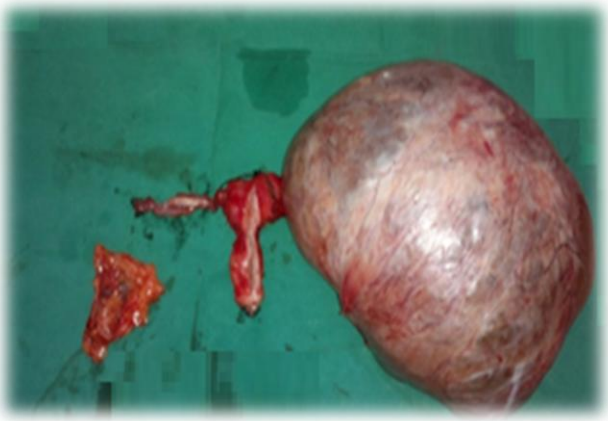


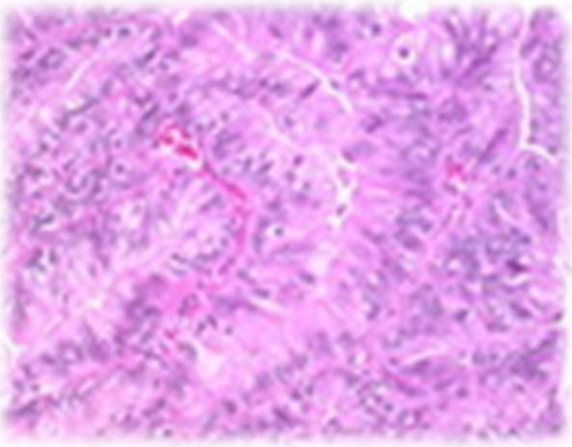
Tall columnar cells with Apical mucin and absence of cilia



Cyst lined by columnar cells with bland basal nuclei and apical mucin

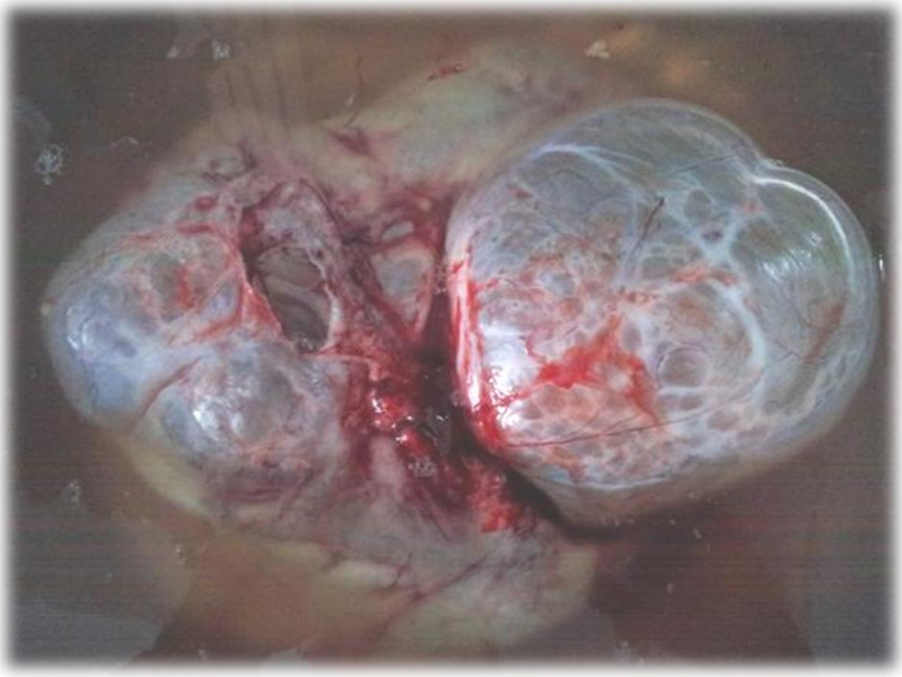
Fig 16.BORDERLINE MUCINOUS OVARIAN TUMOR

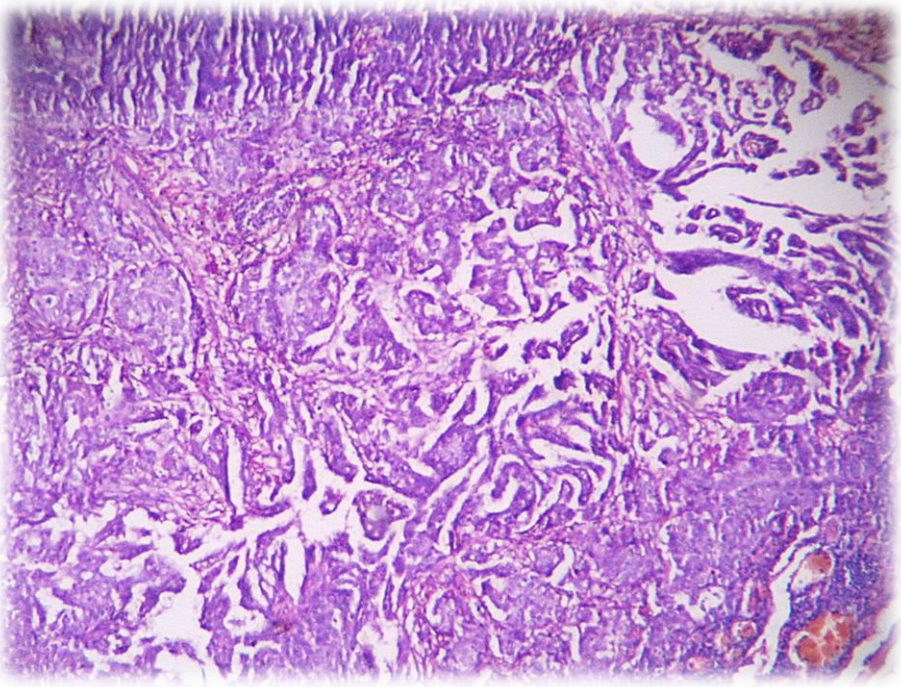




Histopathological examination shows proliferating tall columnar intestinal type of epithelium filled with mucin Nucleus showing hyperchromasia & Stratification with Few mitotic figures

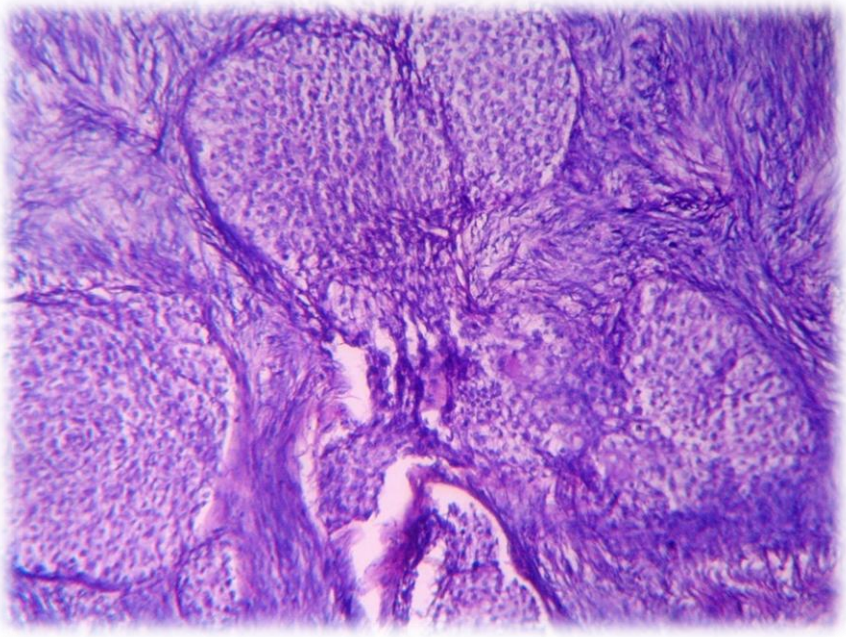
Fig 4.PAPILLARY SEROUS CYST ADENOCARCINOMA



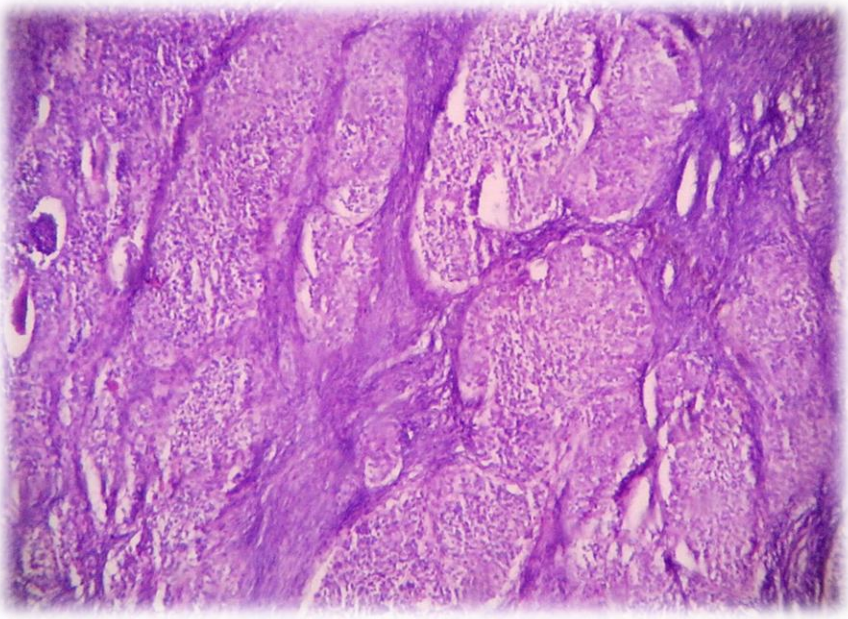


Tumor cells arranged in solid nests and papillary pattern

Fig.8 BILATERAL MALIGNANT BRENNER



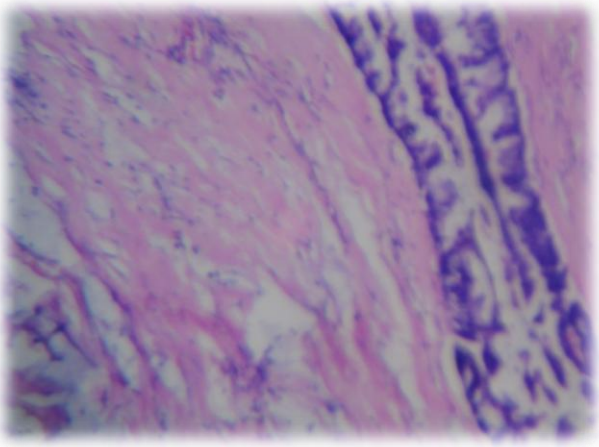
Brenner tumor showing epithelial nests embedded within fibrous stroma



Nests and islands of tumor cells infiltrating the stroma

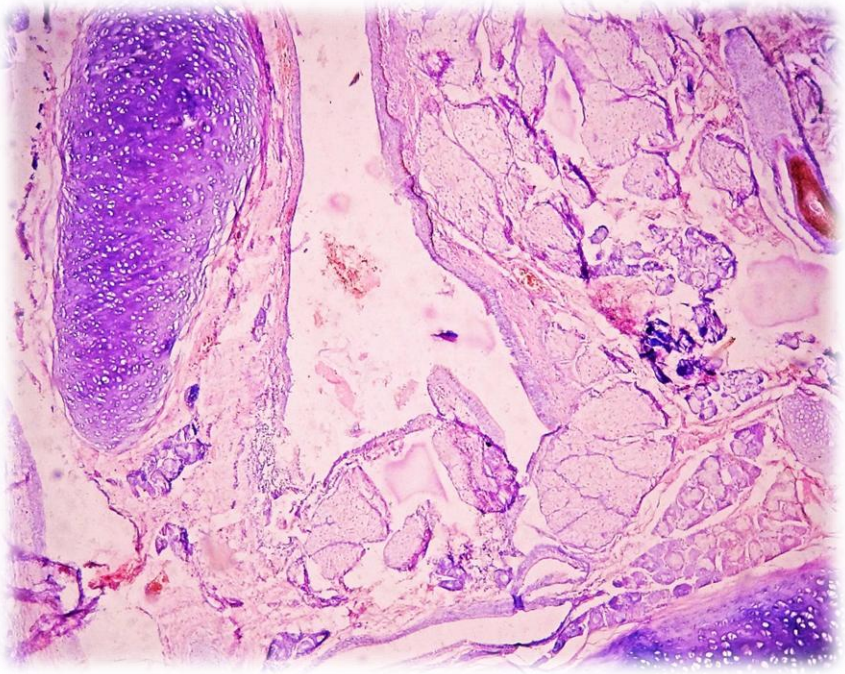
Fig 6.PAPILLARY MUCINOUS CYST ADENOCARCINOMA

Predominantly solid with few mucin containing cystic space



Complex architecture and obvious nuclear atypia

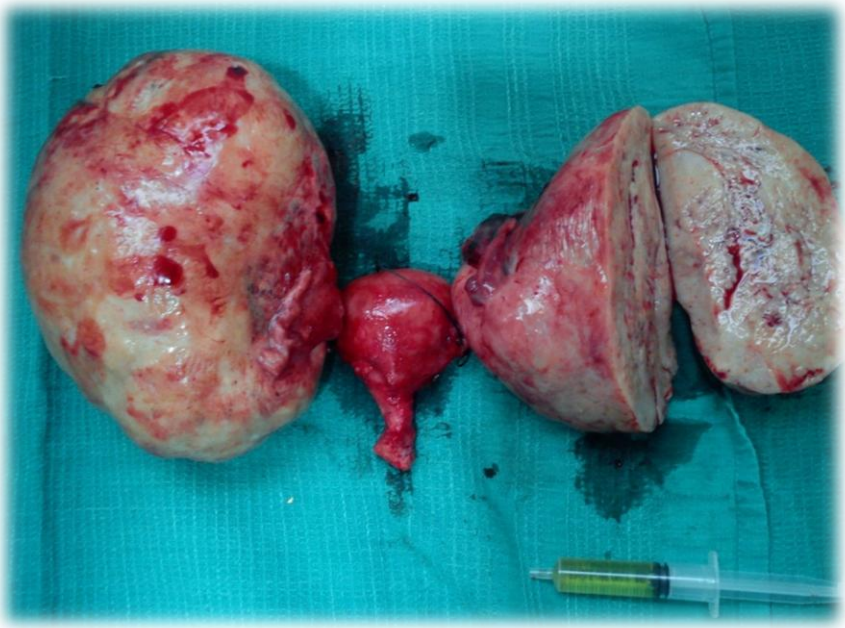
Fig 11.BENIGN CYSTIC TERATOMA

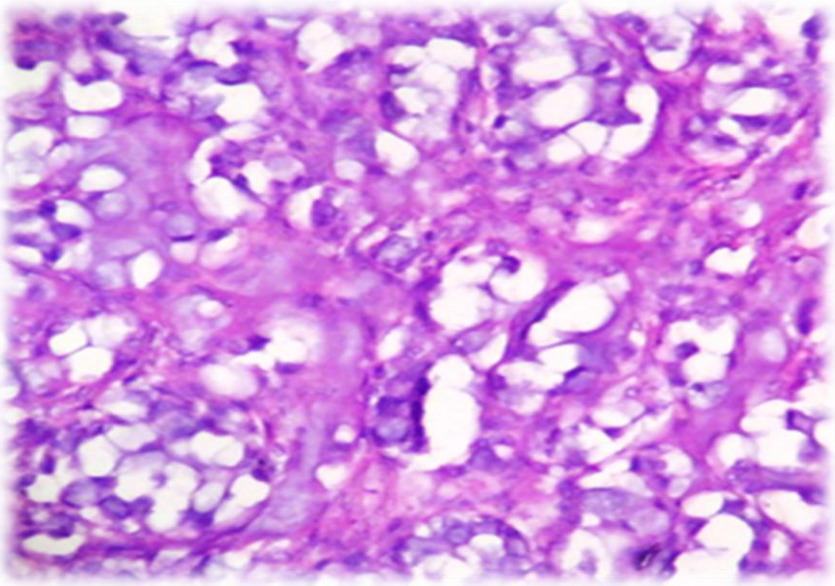


Areas of cartilage, hair and squamous epithelial lining

Sebum, well developed teeth and hair in a mature cystic teratoma

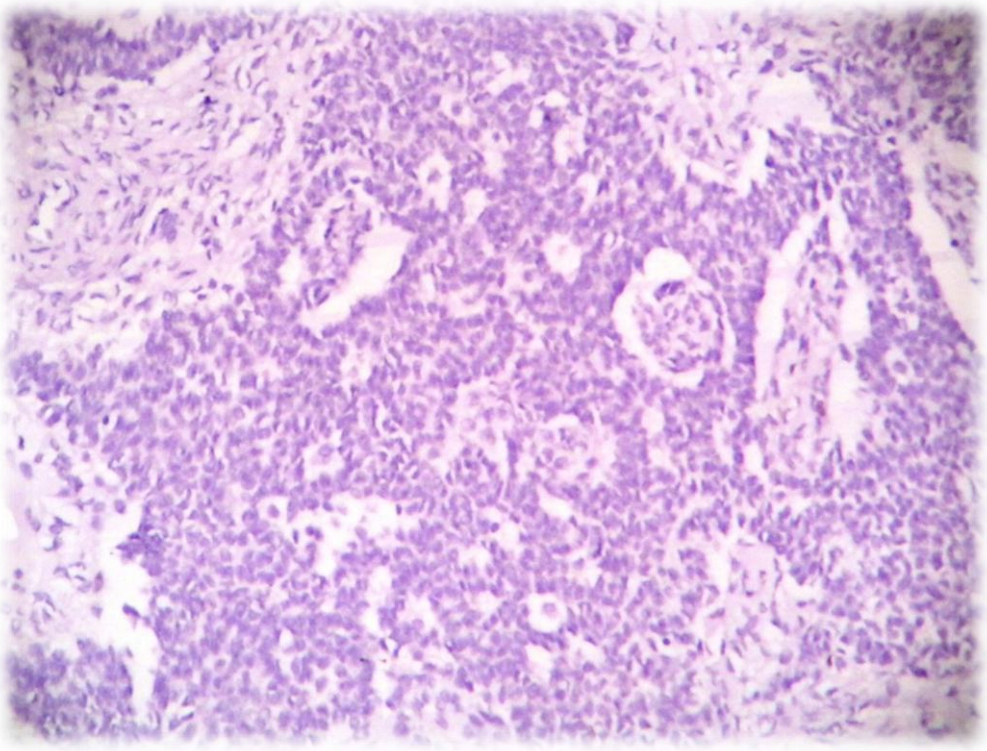
Fig 13.KRUKENBERGS TUMOR

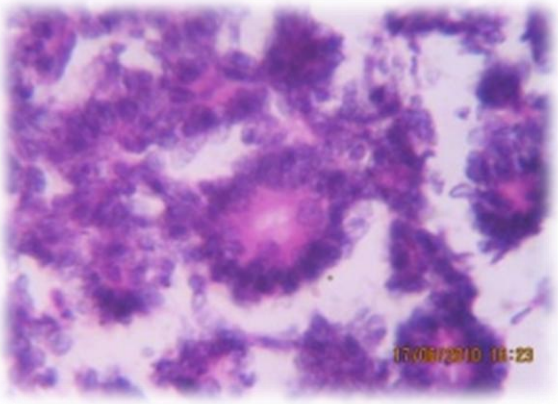




Signet Ring Cells

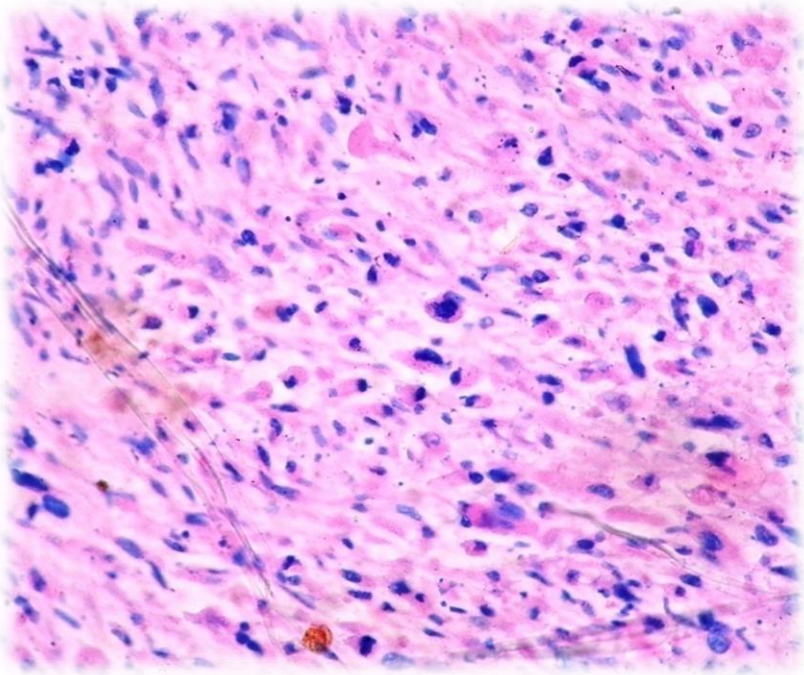
Fig. 9 ADULT GRANULOSA CELL TUMOR





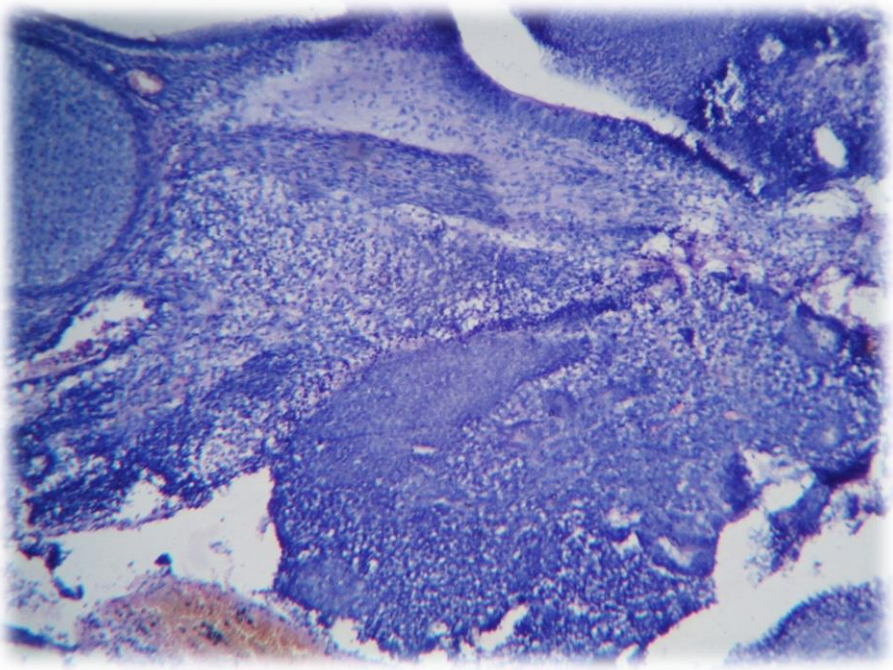
Call Exner bodies

Fig 17.LEIOMYOSARCOMA

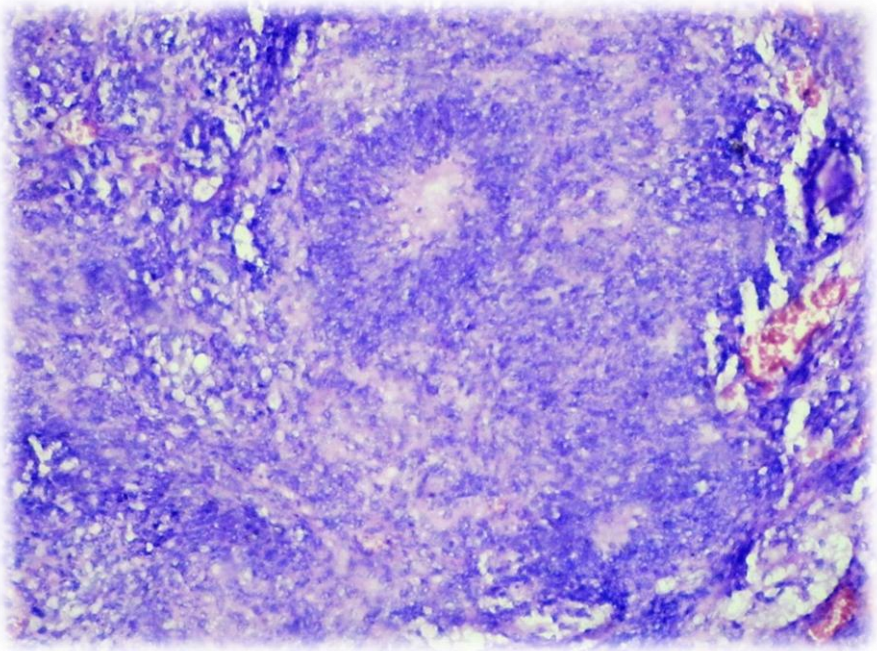


Cells spindle shaped cells with elongated nuclei and eosinophilic cytoplasam of interlacing bundles with whorled appearance pleomorphism, hyperchromatic nuclei with mitoses 14/10 hp

Fig 12.IMMATURE TERATOMA

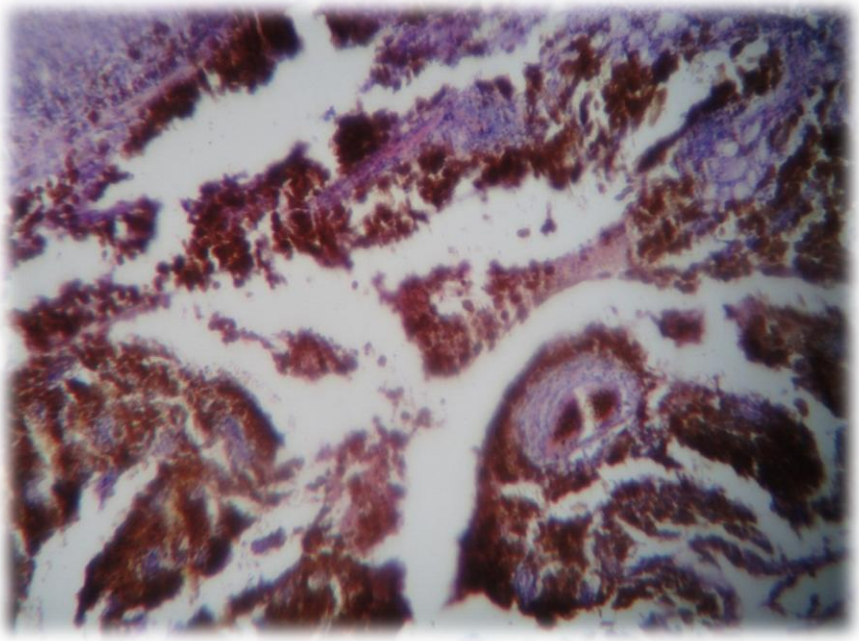


Immature tissue differentiation towards cartilage, glands and bone. Grade II



With Primitive neuroepithelial element

Fig 7.ENDOMETRIOID TUMOR



Well differentiated endometrioid tumor with focal villous architecture

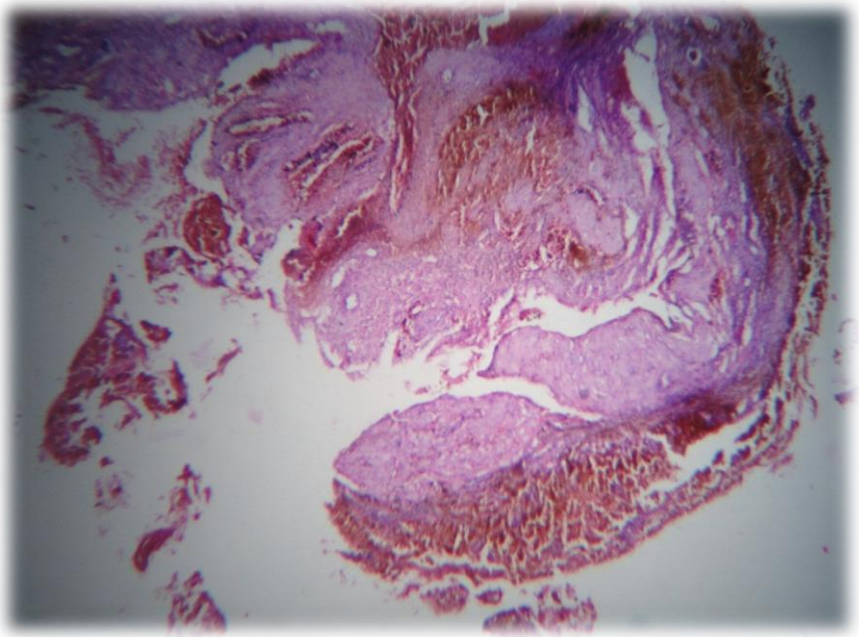
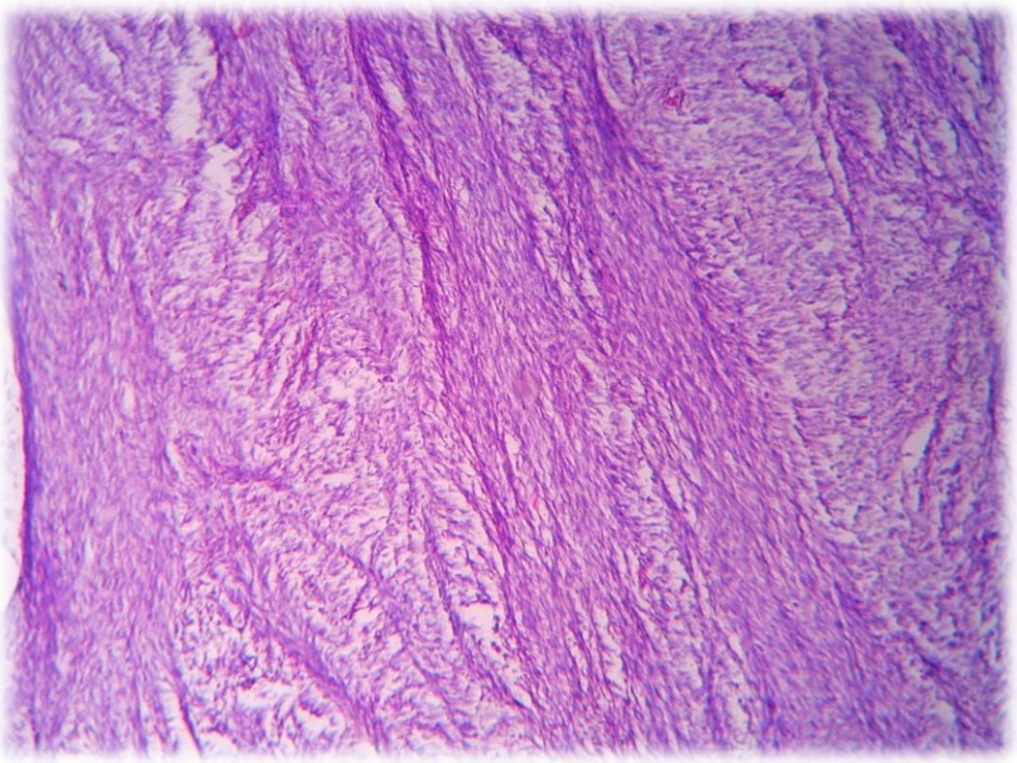


Fig 10.FIBROTHECOMA



Pale lipid containing theca cells merge with spindle cell areas characteristic of fibroma

Fig 14.OVARIAN TUMOR COMPLICATING PREGNANCY

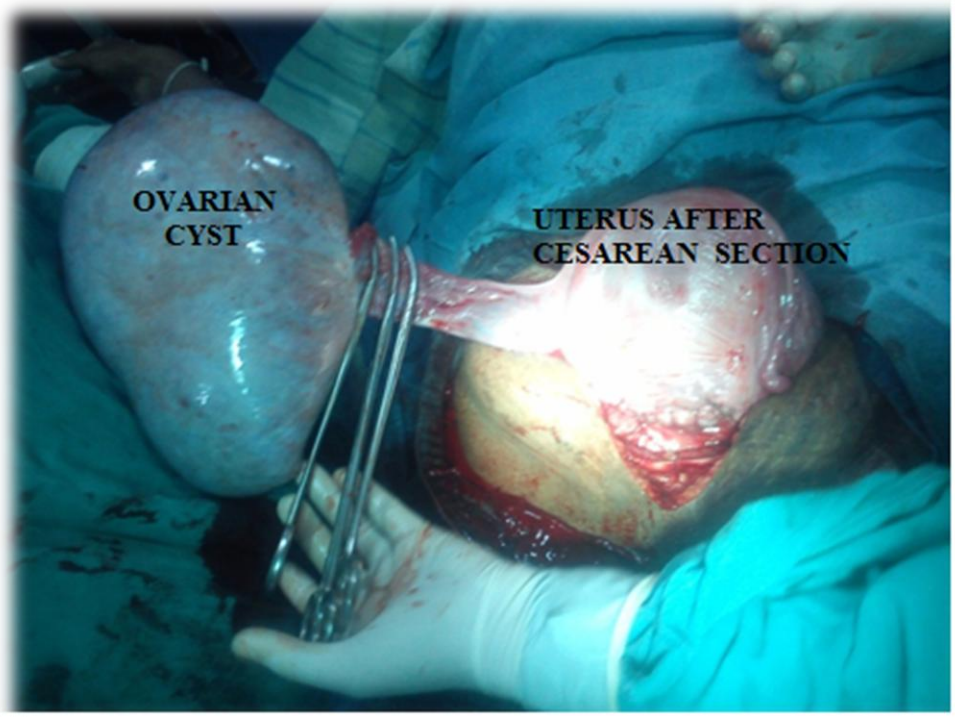


Fig 18a. SMOOTH WALL, SONOLUCENT (SCORE 0)

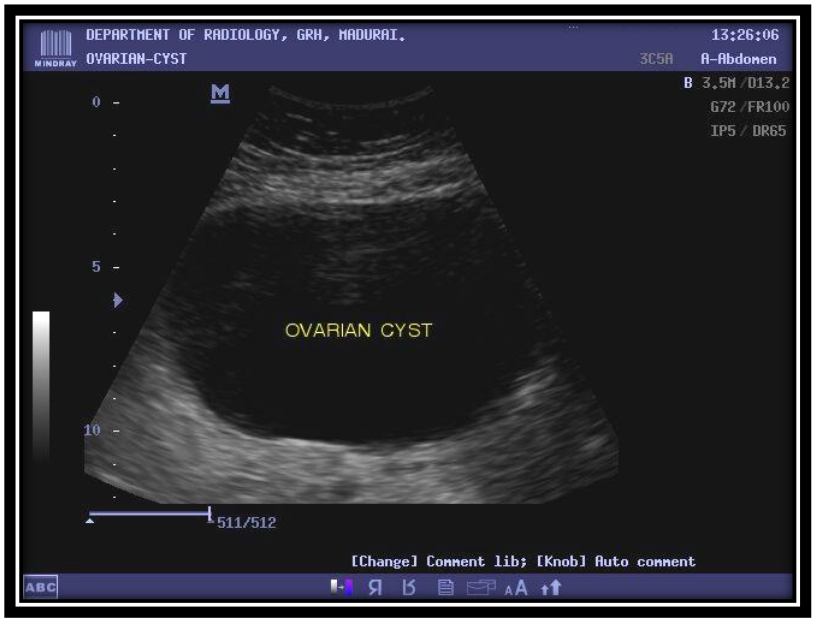


Fig 18b.SMOOTH WALL, DIFFUSE ECHOGENICITY (SCORE 1)

Fig 18c. WALL THICKENING, <3mm FINE SEPTA (SCORE 2)

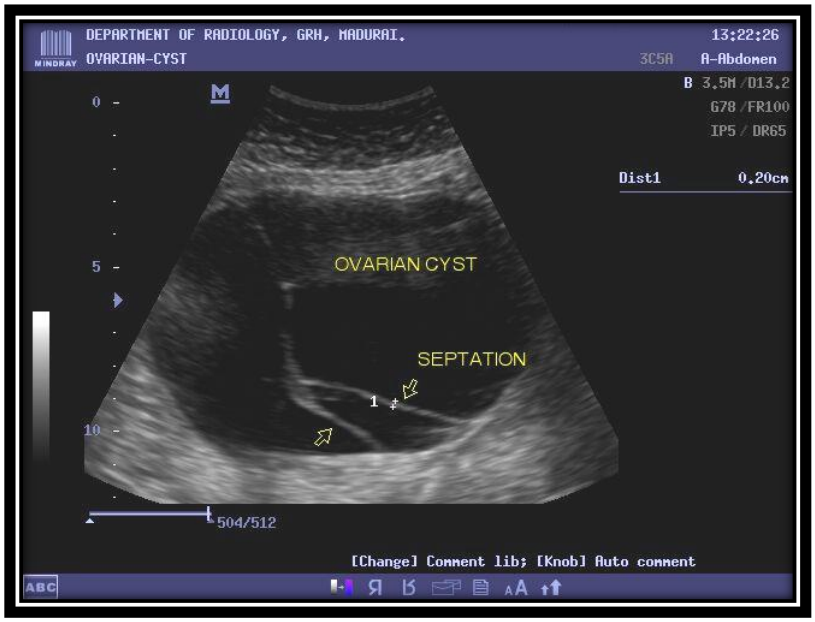


Fig 18d.PAPILLARY PROJECTIONS, SEPTA>3mm (SCORE 3)

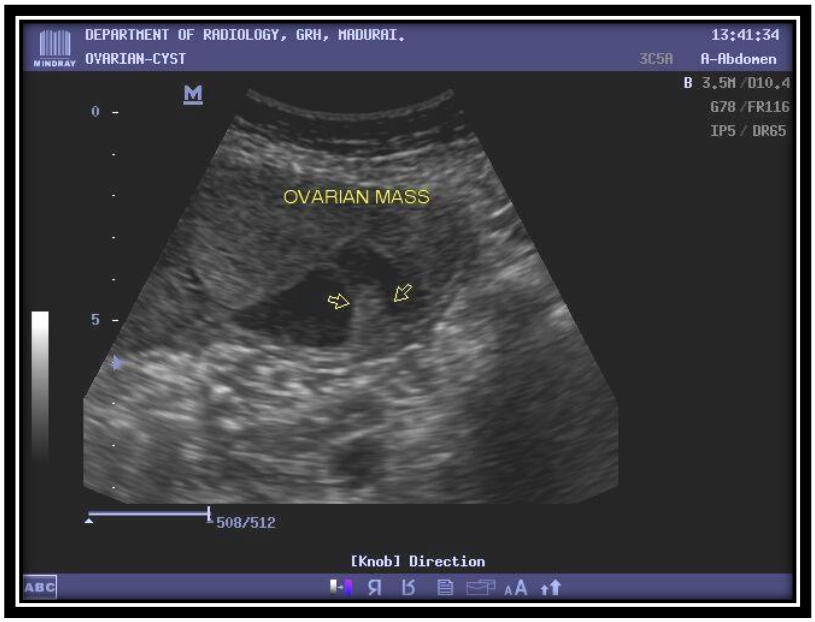


Fig 18e. COMPLEX, PREDOMINANTLY SOLID (SCORE 4)

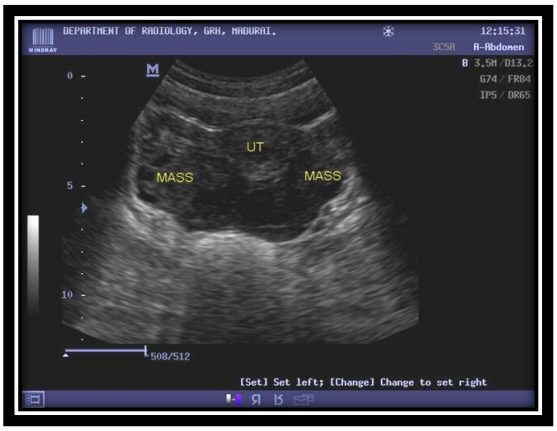


Fig 18f.COMPLEX, SOLID AND CYSTIC AREAS WITH EXTRATUMORAL FLUID (SCORE 5)

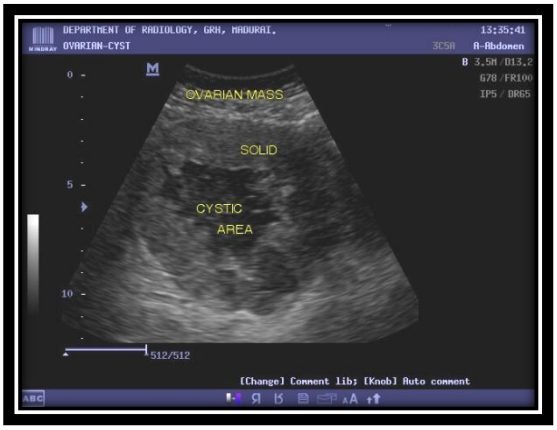


Fig 18g.EXTRATUMORAL FLUID (SCORE 5)

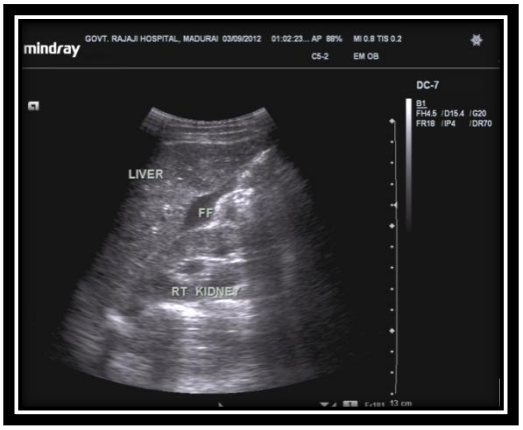


Fig 1. CELLS OF OVARY

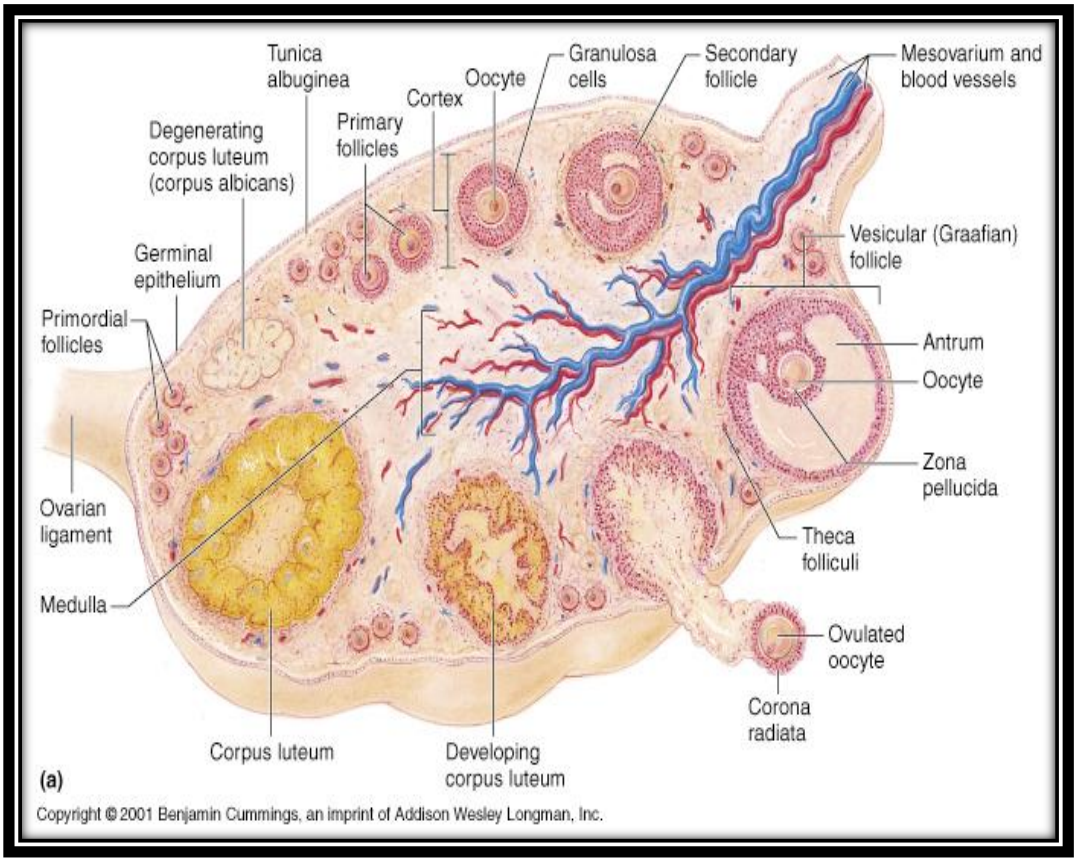


Fig 2. HISTIOGENESIS OF OVARIAN TUMORS

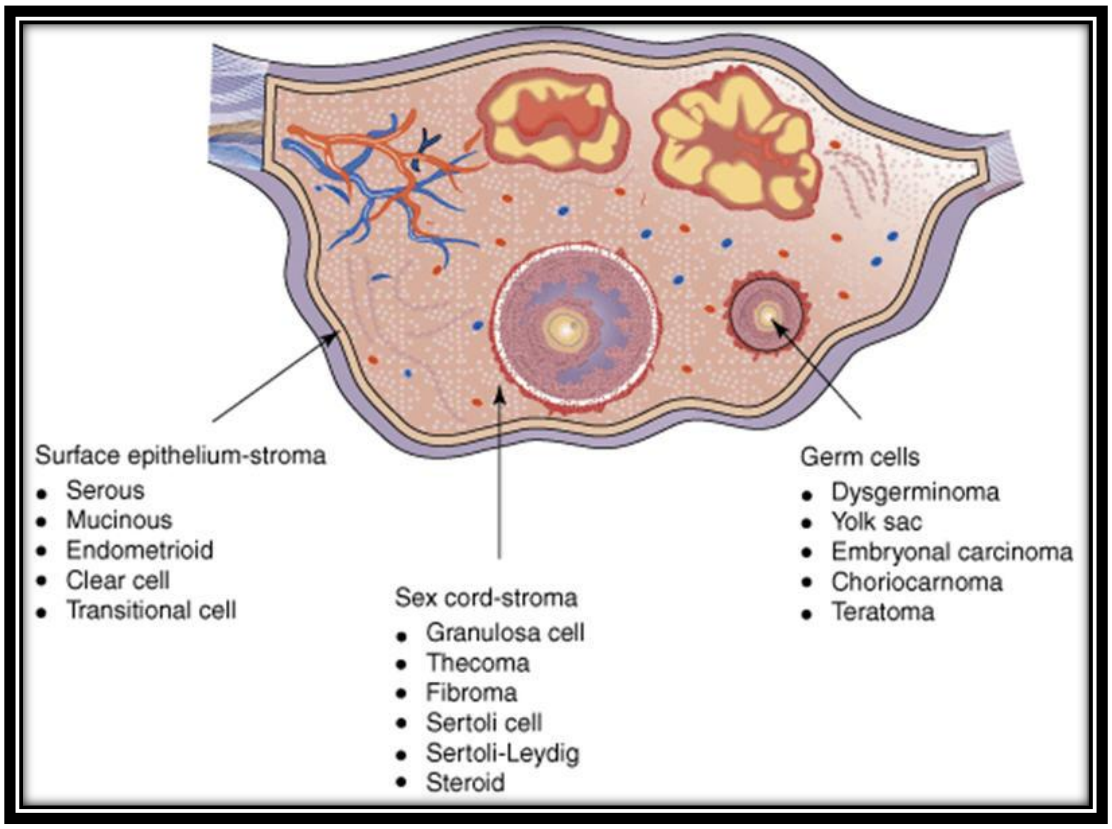
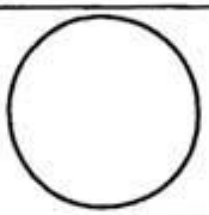
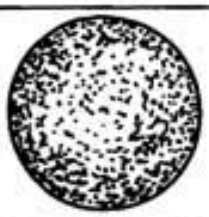
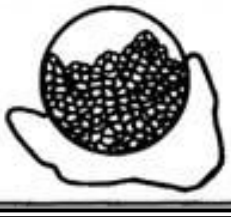


Fig 15. MORPHOLOGICAL SCORING INDEX

	TUMOR VOLUME	TUMOR STRUCTURE
0	<10 cm^3	
1	10-50 cm^3	
2	>50-100 cm^3	
3	>100-200 cm^3	
4	>200-500 cm^3	
5	>500 cm^3	

BIBLIOGRAPHY

1. Schorge, Schaffer, Halvorson: Williams Gynecology: 1st Ed, 2008; 716-753.

2. Berek and Novaks Gynecology: 14th Ed, 2007; 1457-1547.

3. TeLinde's Operative Gynaecology, 10th Ed, Lippincott - Raven, Philadelphia 1997; 1307-1339

4. Campbell S, Bhan V, Royston P: Transabdominal ultrasound screening for early ovarian cancer. Br Med J 299:1363–1367, 1989

5. Granberg S, Wikland M, Jansson I: Macroscopic characterization of ovarian tumors and the relation to the histological diagnosis: Criteria to be used for ultrasound evaluation. GynecolOncol 35:139–144, 1989

6. Sassone AM, Timor-Tritsch IE, Artner A: Transvaginal sonographic characterization of ovarian disease: Evaluation of a new scoring system to predict ovarian malignancy. Obstet Gynecol 78:70–76, 1991

7. DePriest PD, Varner E, Powell J: The efficacy of a sonographic morphology index in identifying ovarian cancer: A multi-institutional investigation. Gynecol Oncol 55:174–178, 1994

8. Lerner JP, Timor-Tritsch IE, Federman A: Transvaginal ultrasonographic characterization of ovarian masses with an improved, weighted scoring system. Am J Obstet Gynecol 170:81–85, 1994

9. Ferrazzi E, Zanetta G, Dordoni D: Transvaginal ultrasonographic characterization of ovarian masses: Comparison of five scoring systems in a multicenter study. Ultrasound Obstet Gynecol 10:192–197, 1997

10. Mol B, Boll D, DeKanter M: Distinguishing the benign and malignant adnexal mass: An external validation of prognostic models. Gynecol Oncol 80:162–167, 2001

11. Ueland FR , DePriest PD , Pavlik EJ , Kryscio RJ , van Nagell JR Jr. Preoperative differentiation of malignant from benign ovarian tumors: the efficacy of morphology indexing and Doppler flow sonography. Gynecol Oncol. 2003 Oct;91(1):46-50.

12. Toshiyuki Hata, Yanagihara, Keiji Hayashi, Yamashir, Yohichi Ohnishi, Akiyama, Atsushi Manabe and Kohji Miyazaki Human Three-dimensional ultrasonographic evaluation of ovarian tumours: a preliminary study Reproduction vol.14 no.3 pp.858–861, 1999

13. J. L. Alcazar, P. Royo, Jurado, M´inguez, Garcia-manero, C. Laparte, R. Galvan and G. Lopez-garcia, Triage for surgical management of ovarian tumors in Asymptomatic women: assessment of an ultrasound-based Scoring system ultrasound obstetgynecol2008; 32: 220–225

14. Shruti Shah, Hishikar V. "Incidence and management of ovarian tumor". Bombay Hospital Journal 2008;50(1):30-33

15. Swamy G.S., Satyanarayana N: Clinicopathological analysis of ovarian tumors; Nepal Med Coll Journal 2010; 12(4);221- 223

16. Williams PL, Dyson M: Gray's anatomy, 37th Ed, London, Churchill Livingstone, 1993.

17. Carolyn Westhoff, Julie A. Britton, Marilie D,. "Oral Contraceptives and Benign Ovarian Tumors" Gammon American Journal of Epidemiology, 2005; Volume152, Issue3, 242-246.

18. Karen H. Lu, Judy E. Garber, "Occult Ovarian Tumors in Women with BRCA1 or BRCA2 Mutations Undergoing Prophylactic Oophorectomy" Journal of Clinical Oncology, Vol 18, Issue 14 (July), 2000: 2728-2732.

19. Charles N. Landen, Jr, Michael J. Birrer, Anil K. Sood, "Early Events in the Pathogenesis of Epithelial Ovarian Cancer", Journal of Clinical Oncology, Vol 26, No 6 (February 20), 2008: pp. 995-1005.

20. Kumar and Cottran Robbins textbook of Pathology, 8th Edition, Chap 22, Pages 1040 – 1052.

21. Fox H and Wells M: Haines and Taylor Obstetrical and Gynaecological pathology, ovarian tumor, 4th Edn New York, Churchill Livingstone, 1996.

22. GezaAcs, MD, PhD, "Serous and Mucinous Borderline (Low Malignant Potential) Tumors of the Ovary", Journal of Clinical Oncology, November 10, 2008 vol. 26 no. 32 5284-5293

23. Vivien W. Chen Ph.D. Bernardo Ruiz M.D., Ph.D. Jeffrey L. Killeen M.D, "Pathology and classification of ovarian tumors" Published 2003 by the American Cancer Society. Volume 97, Issue S10, 15 May 2003: pages 2631–2642.

24. Gobel U, Schneider DT, Calaminus G, "Germ-cell tumors in childhood and adolescence" Annals of Oncology 11: 263-271, 2000.

25. Howkins and Bourne, Shaw's text book of Gynaecology: 2008; 14th Ed 329-349

26. Cunningham, Leveno, Bloom: William's obstetrics :23rd Ed, 2010: 29-30

27. Bakhidze E.V. N. Petrov Ovarian tumours associated with pregnancy Journal of Clinical Oncology, 2006; Vol 24, No 18S (June 20 Supplement), 15063.

28. Jeffcoate's Principles of Gynaecology :2008; 7th Ed,524-560

29. Couto F, Nadkarni NS and Rebello MJP: "Ovarian tumors in Goa: A Clinicopathological study". J obstet and Gynecol India 1993;43(3):408-412.

30. Shahin Rashid, Ghulam Sarwar, Abid Ali ,A Clinico – Pathological Study of Ovarian Cancer in departments of Radiotherapy & Oncology, Sir Ganga Ram Hospital and Mayo Hospital, Lahore. Dec 1998; 13(4):117-125.

31. Malpani AN, Bhattacharya MS, Neuruukar N: Giant ovarian tumor. J Obstet Gynecol India 39(2), 1989: 264-266.

32. DePriest P, Gallion HH, Pavlik EJ: Transvaginal sonography as a screening method for the detection of early ovarian cancer. Gynecol Oncol 65:408–414, 1997

33. Carter J, Lau M, Fowler JM: Blood flow characteristics of ovarian tumors: Implications for ovarian cancer screening. Am J Obstet Gynecol 172:901–907, 1995

34. Fleisher AC, Rodgers WH, Kepple DM: Color Doppler sonography of ovarian masses: A multiparameter analysis. J Ultrasound Med 12:41–48, 1993

35. Roth LM, Anderson MC, and Govan ADT: Sertoli-Leydig cell tumors: A clinico-pathologic study of 34 cases. Cancer 48(1), 1991; 187-197.

KEY TO MASTER CHART

IP NO	:	In patient Number
G/E	:	General physical Examination
P/S	:	Per speculum
P	:	Pain
M	:	Mass
D	:	Distension
U/O	:	Urine output
C	:	Constipation
PM	:	Post-Menopausal
NS	:	Normal Size
Cx, Vg H	:	Cervix & Vagina Healthy
WDPV	:	White discharge per vaginum
IR	:	Irregular Menstrual Irregularity
Nulli	:	Nulligravida
MPV	:	Mass per vaginum
PMB	:	Post Menopausal Bleeding
V	:	Vomiting
Amm	:	Ammenorrhoea
WL	:	Loss of Weight
LA	:	Loss of Appetite

DM	:	Diabetes Mellitus
MA	:	Menstrual Abnormalities
F	:	Fever
PE	:	Pedal Edema
R	:	Regular
NAD	:	No Abnormalities Detected
U	:	Unilateral
L	:	Bilateral
S	:	Smooth wall
T	:	Thickened
So	:	Sonolucent
DE	:	Diffuse echogenicity
ME	:	Mixed echogenicity
TAH	:	Total abdominal hysterectomy
BSO	:	Bilateral salpingo-oophorectomy
RSO	:	Right salpingo-oophorectomy
LSO	:	Left salpingo-oophorectomy
LOC	:	Left ovarian cystectomy
ROC	:	Right ovarian cystectomy
+	:	Present
-	:	Absent

PROFORMA

Name : Marital status :

Age : Ip No :

Date of admission :

Address :

I) Present complaints:

- Mass per abdomen
- Abdominal pain
- Any menstrual irregulartities
- Urinary symptoms
- GIT symptoms
- Edema of lower limbs
- Any discharge per vaginum

II) History of presenting illness

Mass per abdomen

- Onset – insidious/acute
- Pain – present / absent
- Rapidity of growth – rapid / slow

Abdominal pain

- Present/absent
- Onset

- Severity
- associated with nausea/ vomiting
- character of pain / squeezing / coliky / intermittent
- aggravating / relieving factor

Menstrual irregularities

- Menorrhagia – yes/no
- Amenorrhea – yes/no
- Polymenorrhea - yes/no
- Dysmenorrhea - yes/no

Urinary symptoms

- Frequency- yes/no
- Acute retention of urine- yes/no
- Burning micturition- yes/no
- Difficulty in passing urine- yes/no

GIT symptoms

- Nausea
- Vomiting
- Dyspepsia
- Constipation

Edema of lower limbs

- Bilateral/unilateral
- Pitting / non pitting

Any discharge per vagina

- Type / itching / foul smelling

III) Menstrual history

a) Age of menarche – years
b) Past menstrual cycle
    - Regular/irregular
    - Amount of flow- scanty/ moderate/ excessive
    - Dysmenorrhea – yes/no
    - Associated clots - yes/no
c) Attained menopause – yes/no

IV) Obstetric history

a) Parity
b) Last delivery
c) Sterilised/not

V) Past history

a) TB/DM/HTN/Bronchial asthma/any surgeries
b) H/O use of oral contraceptives in the past

VI) Family history

a) TB/DM/HTN/Bronchial asthma/any surgeries
b) Any similar complaints in the family

VII) Personal history

a) Diet – Veg/mixed
b) Appetite – Normal/ decreased
c) Sleep – Normal/ disturbed
d) Bowel – regular / irregular
e) Bladder – Normal/ increased/ decreased

VIII) General physical examination

a. Built / Nourishment
b. Clubbing/pallor/pedal edema
c. Lympadenopathy
d. Breast
e. Temperature – Febrile / afebrile
f. Pulse –
g. BP -

IX) Systemic examination

a) Cardiovascular system
b) Respiratory system
c) Abdominal examination

a. Inspection

a) Shape
b) Movement of quadrants with respiration
c) Mass / swelling

- Size
- Shape
- Extent

d) Any engorged vein
e) Umblicus
f) Hernial sites

b. Palpation

a) Local raise of temperature
b) Tenderness
c) Mass
    - Situation
    - Size
    - Extent
    - Surface
    - Consistency
    - Borders
d) Movements with respiration
e) Any organomegaly

c. Percussion

- Ascities – present / absent

d. Auscultation

- Any bruit- present/absent

X) Per speculum examination

a) Vagina – pale/pink/discharge/rugosity
b) Cervix
    - position – anterior/middle/posterior
    - Erosion – Yes/no
    - Discharge – Yes/ No

XI) Pervaginal examination

- Cervix- consistency/ position/ mobility/tenderness
- Uterus – size/position/ mobility/tenderness
- Mass felt bimanually separate from uterus/ not
- Abdominal mass movement transmitted to cervix/not
- Forniceal examination – full/ free, tender/non tender

XII) Per rectal examination

- Nodulaity
- Rectal wall
- Pouch of douglas

XIII) Investigations

USG abdomen and pelvis

- Bilateral
- Solid/complex
- Multiloculated
- Thickness of cyst wall

- Septal thickness
- Papillary projections.
- Doppler study

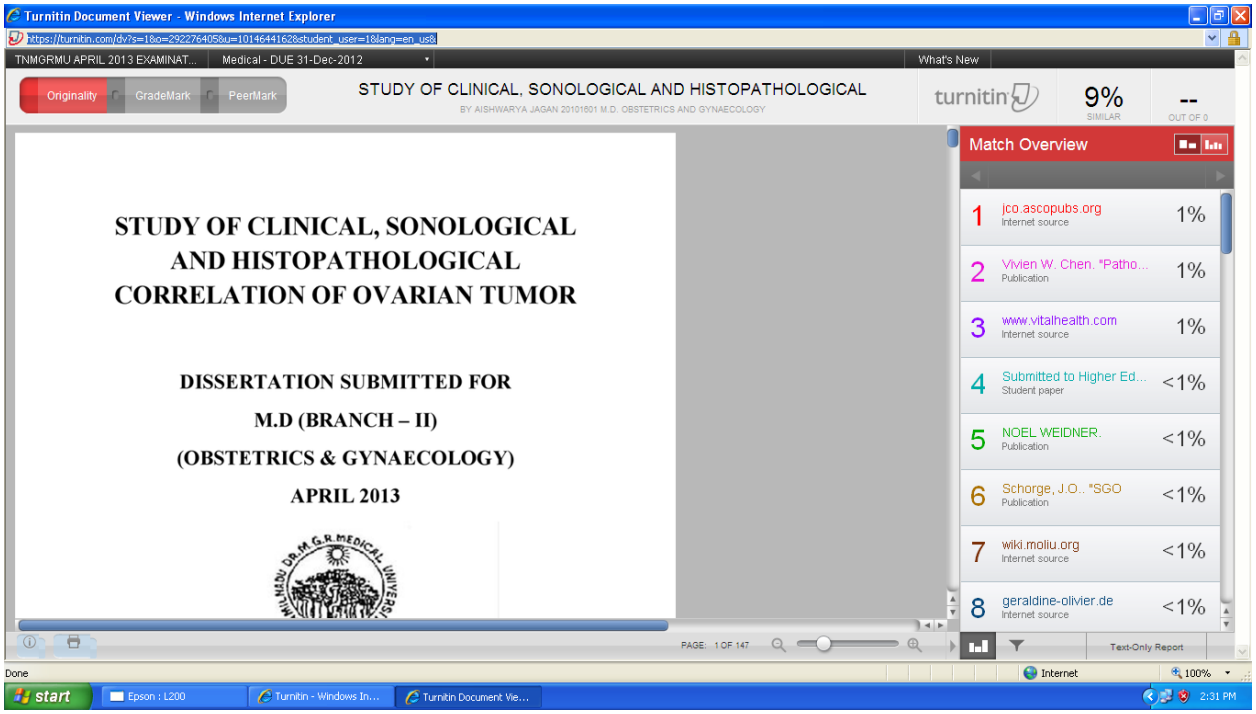
XIV) Treatment

- Surgery
- Peroperative finding

- Tumour - Gross
    - Unilateral/ bilateral
    - Surface – nodular/ smooth
    - Capsule – thickened/ rupture/ hemorrhage
    - Cut section- cystic/ solid
    - Any papillary excrescens

XV) Histopathological examination

Diagnosis:


Turnitin Document Viewer - Windows Internet Explorer
TNMGRMU APRIL 2013 EXAMINAT...
Medical - DUE 31-Dec-2012
What's New
Originality
GradeMark
PeerMark
STUDY OF CLINICAL, SONOLOGICAL AND HISTOPATHOLOGICAL
BY AISHWARYA JAGAN 20101601 M.D. OBSTETRICS AND GYNAECOLOGY
turnitin
9%
SIMILAR
--
OUT OF 0
STUDY OF CLINICAL, SONOLOGICAL
AND HISTOPATHOLOGICAL
CORRELATION OF OVARIAN TUMOR
DISSERTATION SUBMITTED FOR
M.D (BRANCH – II)
(OBSTETRICS & GYNAECOLOGY)
APRIL 2013
Match Overview
1 jco.ascopubs.org Internet source 1%
2 Vivien W. Chen. "Patho... Publication 1%
3 www.vitalhealth.com Internet source 1%
4 Submitted to Higher Ed... Student paper <1%
5 NOEL WEIDNER. Publication <1%
6 Schorge, J.O.. "SGO Publication <1%
7 wiki.moliu.org Internet source <1%
8 geraldine-olivier.de Internet source <1%
PAGE: 1 OF 147
Text-Only Report
Done
Internet
100%
start
Epson : L200
Turnitin - Windows In...
Turnitin Document Vie...
2:31 PM

Ref. No. 3104/E4/3/2012

Govt.Rajaji Hospital,Madurai.20.
Dated: .03.2012

Institutional Review Board / Independent Ethics Committee.
Dr. A. Edwin Joe, M.D (FM), BL.,
Dean, Madurai Medical College & 2521021 (Secy)
Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 29.03.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5. Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6. Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7. Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8. Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9. Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10. Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Aishwarya Jayam	PG, M.D (ob gyn)	Clinical study of ovarian tumours	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

DEAN

To
All the above members and Head of the Departments concerned.
All the Applicants.

S.No	NAME	AGE	I.P.NO.	MODE OF PRSENTATION	MENSTRuAL HISTORY	PARITY	G/E	PER ABDOMEN					PER SPECULUM	PER VAGINAL		USG										LATERALITY	SURGERY	HPE
								SIZE	CONSISTENCY	MOBILITY	TENDERNESS	ASCITES		UTERUS	MASS	SIZE	VOLUME	TUMOR WALL	ECHOGENICITY	PAPILLARY PROJECTIONS	COMPONENTS	ASCITES	VOLUME SCORE	STRUCTURAL SCORE	TOTAL MORPHOLOGICAL SCORE			
1	Sundari	55	13232	P	PM	P6L6	NAD	6x8	Cystic	+	+	-	Cx,Vg H	Atropic	+	2.4x4x6.8	34.14	S	So	-	Cystic	-	1	0	1	U	TAH BSO	Benign Serous Cystadenoma
2	Bhuavaneshwari	32	13751	P	R	P2L2	NAD	10x8	Cystic	+	+	-	Cx,Vg H	NS	+	9.2x5.1x6.2	152.1	S	So	-	Cystic	-	2	0	2	U	RO	Benign Serous Cystadenoma
3	Pandiammal	55	15626	P, M, D	IR	P3L3	NAD	12x10	Cystic	+	+	-	Cx,Vg H	NS	+	9x12x5.4	305.01	S	So	+	Cystic	-	4	0	4	U	TAH BSO	Benign Serous Cyatadenoma
4	Rajathi	29	15188	P,M	R	Nulli	NAD	12x12	Cystic	+	-	-	Cx,Vg H	NS	+	12.7 x 10.8x11.2	803.4	S	So	-	Cystic	-	5	1	6	U	RO	Benign Mucinous Cystadenoma
5	Gundhammal	75	16724	P,D	PM	P6L6	CAC	30x30	Cystic	+	-	-	Cx,Vg H	Atropic	+	29.5x24x21.6	7998.1	S	DE	-	Cystic	-	5	1	6	U	TAH BSO ICO	Benign Mucinous Cystadenoma
6	Mahamani	45	18044	P,u/O, C	PM	P5L2	NAD	-	-	-	-	-	Cx,Vg H	NS	+	2.1x3.9x4	17.13	S	So	-	Cystic	-	1	0	1	U	LOC	Benign Serous Cyatadenoma
7	Suseela	30	17626	P	R	P2L2	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x5x7.5	196.12	S	So	-	Cystic	-	3	0	3	U	LOC	Benign Serous Cystadenoma
8	Deivanai	35	1809	P	IR	Nulli	NAD	10x10	Cystic	+	-	+	Cx,Vg H	Bulky	+	9.8x10x6	307.5	S	So	-	Cystic	*	4	5	9	U	LOC IOC	Benign Serous Cystadenoma
9	Alagu	45	18550	P	PM	P3L3	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10x14.5x12	910.02	S	So	-	Cystic	-	5	0	5	U	TAH BSO	Benign Mucinous Cystadenoma
10	Fathima	28	20992	P, D	IR	P2L2	NAD	20x20	Firm	+	+	-	Cx,Vg H	NS	+	22.5x20x13.5	3177.2	T	DE	+	Complex	-	5	4	9	U	TAH BSO	Granulosa Cell Tumor
11	Saroja	40	20128	P,WL,LA	R	P1L1	NAD	8x8	Cystic	+	-	-	Cx,Vg H	NS	+	8.2x6.8x5	145.8	S	DE	-	Cystic	-	3	2	5	U	TAH BSO ICO	Benign Serous Cystadenoma
12	Chinnapillai	38	21976	P, DM	R	P5L5	NAD	15x10	Cystic	+	-	-	Cx,Vg H	NS	+	14x12x7	615.04	S	So	-	Cystic	-	5	0	5	U	RO	Benign Mucinous Cystadenoma
13	Dharmaboopathi	60	21058	P, C	PM	P3L3	OBS	30x25	Hard	+	-	-	Cx,Vg H	Atropic	+	30.2x28.1x22.5	9986.13	T	ME	+	Complex	-	5	4	9	B	TAH BSO ICO	Krukenberg tumor
14	Rajathi	25	1607	P	R	P1L1	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x8x5.2	217.56	S	DE	+	Cystic	-	3	3	6	U	ROC	Benign Cystic Teratoma
15	Susila	40	24635	M	IR	P1L1	NAD	25x25	Cystic	+	-	-	Cx,Vg H	Bulky	+	30x21.5x18	6072.03	T	DE	+	Complex	-	5	4	9	U	TAH BSO ICO	Granulosa Cell Tumor
16	Indirani	51	22634	WD, WL	PM	P4L4	NAD	15x15	Hard	+	-	+	Cx,Vg H	Atropic	+	13.5x14x10.2	1008.2	T	ME	+	Complex	+	5	5	10	B	TAH BSO ICO	Papillary Serous Cystadenocarcinoma

17	Sumitha	38	24134	M	R	P1L1	NAD	18x15	Hard	+	-	+	Cx,Vg H	NS	+	14.8x20x18.5	2863.9	T	DE	-	Complex	+	5	5	10	U	TAH BSO ICO	Adenocarcinoma
18	Selvi	30	1487	P,WD, V,LA	R	P3L3	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10x 6x5	156.9	S	So	-	Cystic	-	3	0	3	U	LOC	Benign Serous Cystadenoma
19	Alagammal	50	2508	D	PM	P4L3	NAD	30x30	Cystic	+	-	-	Cx,Vg H	Atropic	+	18x12.8x30	3614.9	T	ME	+	Complex	-	5	4	9	U	TAH BSO ICO	Benign Mucinous Cystadenoma
20	Periyakka	50	4981	P,D	R	P1L1	NAD	13x12	Hard	+	-	+	Cx,Vg H	NS	+	10.5x9.8x11	591.9	T	ME	+	Complex	+	3	5	8	B	TAH BSO ICO	Mucinous Cystadenocarcinoma
21	Mahalakshmi	20	4986	P,D	R	P2L0	CAC	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10.3x8x4.7	202.5	S	DE	-	Cystic	-	4	1	5	U	TAH BSO ICO	Ovarian Fibrothecoma
22	Sara	35	7125	P	R	P3L3	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10.5x7.2x5	32.9	T	So	-	Cystic	-	1	2	3	U	LO	Benign Serous Cystadenoma
23	Mariyam Beevi	40	7107	P	PM	Nulli	NAD	-	-	-	-	-	Cx,Vg H	NS	+	4.9x 3.1 x 5	39.7	S	So	-	Cystic	-	1	0	1	B	TAH BSO	Benign Serous Cystadenoma
24	Sankarammal	60	5113	D	PM	P1L1	NAD	15x15	Hard	+	-	+	Cx,Vg H	Atropic	+	12.5x14x11.5	1052.5	T	ME	-	Complex	+	5	5	10	U	TAH BSO ICO	Mucinous Cystadenocarcinoma
25	Nagalakshmi	35	9268	P	R	Nulli	NAD	15x10	Firm	+	-	-	Cx,Vg H	NS	+	15x12.5x13	1274.81	T	ME	+	Complex	-	5	4	9	U	Lap omental Biopey	Poorly Differentiated Carcinoma
26	Selvi	42	10354	D	R	P4L3	NAD	20x20	Cystic	+	-	-	Cx,Vg H	NS	+	15x12.5x13	1274.8	S	So	-	Cystic	-	5	0	5	U	TAH BSO	Benign Serous Cystadenoma
27	Lourdhmary	38	12571	D	R	P2L2	NAD	10x15	Cystic	+	-	-	Cx,Vg H	NS	+	10.2x9.1x6	291.26	S	So	-	Cystic	-	4	0	4	U	TAH BSO	Benign Serous Cystadenoma
28	SubaithaBegam	40	11904	LA	R	Nulli	NAD	20x20	Cystic	+	-	-	Cx,Vg H	NS	+	18.5x12x8.2	952	S	So	-	Cystic	-	5	0	5	U	TAH BSO	Benign Mucinous Cystadenoma
29	Bakayalakshmi	55	13085	P	PM	P5L5	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10x6x8.5	266.73	S	DE	-	Cystic	-	4	1	5	U	TAH BSO	Benign Serous Cystadenoma
30	Petchiyammal	40	39417	P	R	P3L3	NAD	-	-	-	-	-	Cx,Vg H	NS	+	3.4x4.2x5.8	43.31	S	So	-	Cystic	-	2	0	2	U	TAH BSO	Benign Serous Cystadenoma
31	Valli	40	15765	P	R	P3L3	NAD	16x20	Variable	-	+	+	Cx,Vg H	NS	*	16.5x15x12	1553.31	T	ME	+	Complex	.	5	4	9	U	TAH BSO ICO	Mucinous Cystadenocarcinoma
32	Pandiselvi	21	8803	P	R	Nulli	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	8x5x6	125.52	S	So	-	Cystic	-	3	0	3	U	LOC	Benign Serous Cyatadenoma
33	SajethaBegam	40	7226	M	R	P3L3	NAD	20x20	Firm	+	+	-	Cx,Vg H	NS	+	20x25x17.5	4576.2	S	DE	-	Cystic	-	5	1	6	U	TAH BSO ICO	Leiomyosarcoma
34	Panchavarnam	26	22694	P	R	Nulli	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10x8 x7	292.88	S	So	-	Cystic	-	4	0	4	U	LOC	Benign Serous Cystadenoma
35	Selvi	24	2025	P	R	P2L2	NAD	9x10	Cystic	+	-	-	Cx,Vg H	NS	+	9x7x6.5	214.16	S	ME	-	Cystic	-	4	1	5	U	RO	Benign Cystic Teratoma
36	Shajitha	26	4038	P,V	R	P2L2	NAD	10x12	Cystic	+	-	-	Cx,Vg H	NS	+	10.2x9.5x8	405.42	S	So	-	Cystic	-	5	0	5	U	RO	Benign Serous Cystadenoma
37	Rakkammal	58	7236	P,D	R	P3L3	NAD	20x20	Hard	+	-	+	Cx,Vg H	NS	+	18.8x15x13.2	1946.8	T	ME	-	Complex	+	5	4	9	B	TAH BSO ICO	Mucinous Cystadenocarcinoma

38	Veeralakshmi	47	9939	D,LA	R	P2L2	NAD	20x20	Cystic	+	-	-	Cx,Vg H	NS	+	16.5x15x12	1553.31	S	So	+	Cystic	+	5	5	10	U	TAH BSO ICO	Benign Serous Cystadenoma
39	Guruvammal	60	9354	P,LA,D	PM	P3L3	NAD	20x18	Variable	+	-	-	Cx,Vg H	Atropic	+	10.5x8x11	483.25	T	ME	+	Complex	-	4	4	8	B	TAH BSO ICO	Poorly Differentiated Papillary Carcinoma
40	Veeramuthu	17	12038	MA	IR	Nulli	NAD	8x6	Cystic	+	+	-	Cx,Vg H	NS	+	8x6x5	125.52	S	So	-	Cystic	-	3	0	3	U	LOC	Benign Papillary Serous Cystadenoma
41	Malarvizhi	20	13178	P	R	Nulli	NAD	20x18	Variable	+	+	+	Cx,Vg H	NS	+	21.5x16x12	2158.94	T	DE	+	Complex	+	5	5	10	U	LOC ICO	Mucinous Cystadenocarcinoma
42	Rajeshwari	23	15308	P	R	Primi	NAD	15x10	Hard	+	-	-	Cx,Vg H	NS	+	15x10x11	862.95	S	So	+	Complex	-	5	4	9	U	LO	Benign Mucinous Cystadenoma
43	Rahmath nisha	55	15861	P	PM	P1L1	NAD	-	-	-	-	-	Cx,Vg H	Atropic	+	2x1.2x3	3.76	S	So	-	Cystic	-	0	0	0	U	TAH BSO ICO	Benign Serous Cystadenoma
44	Shanthi	30	14373	P	R	P3L3	NAD	-	-	-	-	-	Cx,Vg H	NS	+	3.2x4.8x3	21.09	S	So	-	Cystic	-	1	0	1	U	LOC	Benign Serous Cyatadenoma
45	Ammapillai	65	15320	P	PM	P4L4	NAD	10x8	Cystic	+	-	-	Cx,Vg H	Atropic	+	10x8.2x5	214.43	S	So	-	Cystic	-	4	0	4	U	TAH BSO	Benign Serous Cyatadenoma
46	Sundareshwari	35	15786	P	R	P14	NAD	9x8	Cystic	+	-	-	Cx,Vg H	NS	+	9x5x6	141.21	S	So	-	Cystic	-	3	0	3	U	RO	Benign Serous Cystadenoma
47	Subbulakshmi	30	16799	P,D,WL,F	R	P2L2	NAD	15x18	Cystic	+	-	-	Cx,Vg H	NS	+	18x16.5x12	1863.97	S	ME	-	Complex	-	5	3	8	U	TAH BSO ICO	Benign Mucinous Cystadenoma
48	Chellam	30	17277	P	R	Nulli	NAD	10x7	Cystic	+	-	-	Cx,Vg H	NS	+	9x6x5.2	146.85	S	So	-	Cystic	-	3	0	3	U	ROC	Benign Serous Cystadenoma
49	Sathya Bama	38	17754	P	R	P2L2	NAD	8x6	Cystic	+	-	-	Cx,Vg H	NS	+	8.6x6x4	107.94	S	DE	-	Cystic	-	3	1	4	U	RSO	Benign Serous Cystadenoma
50	Rajam	55	18252	P	R	P6L6	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	9x8x7.5	282.42	T	ME	-	Cystic	-	5	2	7	U	TAH BSO	Benign Mucinous Cystadenoma
51	Jeyanthi	21	18756	P	R	Nulli	NAD	14x15	Cystic	+	-	-	Cx,Vg H	NS	+	12x13x11.5	938.26	S	DE	-	Cystic	-	5	1	6	U	LO	Benign Endometriotic Tumor
52	Valli	60	25253	P	R	P2L2	NAD	20x20	Hard	+	+	+	Cx,Vg H	NS	+	18x16.5x12	1863.97	T	ME	+	Complex	+	5	5	10	U	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
53	Murugeshwari	22	3576	P	R	G2P1L1	NAD	-	-	-	-	-	Cx,Vg H	NS	+	8x5x4.5	94.14	S	So	-	Cystic	-	3	0	3	U	LO	Benign Serous Cyatadenoma
54	Serumathy	28	2915	P,V	R	P1L1	NAD	10x15	Cystic	+	-	-	Cx,Vg H	NS	+	10x8x6.5	271.96	S	ME	-	Cystic	-	4	1	5	U	LO	Benign Cystic Teratoma
55	Vasantha	70	11286	P	R	P5L5	NAD	20x22	Hard	+	+	-	Cx,Vg H	NS	+	25x22x18.5	5321.5	S	So	-	Cystic	-	5	0	5	B	BSO	Papillary Serous Cystadenocarcinoma
56	Chandra	38	4629	P,D	R	P2L2	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10x8 5x5	222.27	T	DE	-	Cystic	-	4	2	6	B	TAH BSO	Serous cystadenocarcinoma
57	Rathinam	45	4146	P	R	P4L4	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x8x6.5	271.96	S	So	-	Cystic	-	4	0	4	U	TAH BSO	Benign Serous Cystadenoma
58	Muthu	35	7806	P	R	P5L5	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x8x7.2	301.24	S	So	-	Cystic	-	4	0	4	U	LO	Benign Serous Cystadenoma

59	Vanmathi	17	8368	P	R	Nulli	Anemic	20x25	Hard	+	+	-	Cx,Vg H	NS	+	21x18x14.2	2807.25	T	DE	+	Complex	-	5	4	9	U	LO ICO	Immature Teratoma
60	Kamatchi	40	7799	P,D	R	P3L2	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x6x4.5	141.21	S	So	-	Cystic	-	3	0	3	U	RO	Benign Mucinous Cystadenoma
61	Ariyanatchi	50	11050	P,D	R	P4L4	NAD	15x17	Hard	+	+	+	Cx,Vg H	NS	+	20x18x15.5	2918.34	T	So	+	Cystic	+	5	3	8	B	TAH BSO ICO	Serous Cystadenocarcinoma
62	Meenatchi	26	12128	D	R	P2L2	NAD	.	.	.	.	-	Cx,Vg H	NS	+	6x4x3.5	43.93	S	So	-	Cystic	.	1	0	1	U	LO	Benign Serous Cystadenoma
63	Parameshwari	55	5572	P,D	R	P1L1	NAD	10X15	Cystic	+	+	-	Cx,Vg H	NS	+	9.2x5x7	168.4	S	So	-	Cystic	.	3	0	3	U	TAH BSO	Benign Serous Cystadenoma
64	Ganesammal	35	12745	P	R	P2L2	NAD	10x12	Cystic	+	.	.	Cx,Vg H	NS	+	10x12.5x9	588.37	S	So	.	Cystic	.	5	0	5	U	TAH RSO	Benign Mucinous Cystadenoma
65	Rani	42	13843	P,MA	IR	P4L2	Anemic	15x20	Variable	+	+	+	Cx,Vg H	Bulky	+	14.5x13x16	1577.368	T	ME	+	Complex	.	5	4	9	U	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
66	Mohana	54	47472	P,D	R	P2L2	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	9x7x6.5	214.16	S	So	-	Cystic	-	4	0	4	U	LOC	Benign Serous Cystadenoma
67	Thangammal	55	16473	PMB	PM	P4L3	NAD	6x8	Cystic	+	-	-	Cx,Vg H	Atropic	+	6x5x3.5	54.91	S	DE	-	Cystic	-	2	1	3	B	TAH BSO ICO	Benign Papillary Serous Cystadenofibroma
68	Nagalakshmi	26	17459	Asymp	R	A1	NAD	9x8	Cystic	+	-	-	Cx,Vg H	NS	+	9x8x4.5	169.45	S	ME	-	Cystic	-	3	1	4	U	LO	Benign Cystic Teratoma
69	Sulochana	35	2363	P	R	P2L2	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10x7x5.2	190.37	S	So	-	Cystic	-	3	0	3	U	LO	Benign Serous Cystadenoma
70	Muthulakshmi	20	2855	P	R	Nulli	NAD	9x8	Cystic	+	-	-	Cx,Vg H	NS	+	9.5x4x6.2	123.2	S	So	-	Cystic	-	3	0	3	U	ROC	Benign Serous Cystadenoma
71	Shenbagavalli	42	2857	P	R	P3L3	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10.2x7.2x6	230.45	S	So	-	Cystic	-	4	0	4	U	TAH BSO	Benign Serous Cystadenoma
72	Devi	47	7017	P,D	R	P3L3	NAD	20x25	Variable	-	-	+	Cx,Vg H	NS	+	20x15.2x12	1907.9	T	ME	+	Complex	+	5	5	10	U	TAH BSO	Serous Cystadenocarcinoma
73	Mathani	24	11270	P	R	P2L2	NAD	10x15	Cystic	+	-	-	Cx,Vg H	NS	+	10.8x7.4x8	334.38	S	So	-	Cystic	-	4	0	4	U	ROC	Benign Mucinous Cystadenoma
74	Chinnammal	60	12442	P	PM	P7L5	NAD	-	Cystic	+	-	-	Cx,Vg H	Atropic	+	3x4.2x3.2	21.08	T	So	+	Cystic	-	1	3	4	B	TAH BSO	Papillary Serous Cystadenocarcinoma
75	Murugeshwari	39	12961	P	R	P3L3	NAD	-	Variable	+	+	-	Cx,Vg H	NS	+	5x5.6x4	58.57	T	So	-	Cystic	-	2	2	4	B	TAH BSO ICO	Adenocarcinoma
76	Rakku	50	14123	P	PM	P2L2	NAD	10x10	Variable	+	+	-	Cx,Vg H	NS	+	9.8x10x9	461.28	T	So	+	Cystic	-	4	3	7	B	TAH BSO ICO	Adenocarcinoma
77	Ezuvakka	52	1670	P	R	P1L1	NAD	10x10	Firm	+	-	-	Cx,Vg H	NS	+	9x10x8.2	385.974	S	So	-	Cystic	-	4	0	4	U	TAH BSO	Papillary Serous Cystadenocarcinoma
78	Bakkiyam	63	13467	P	PM	P6L5	NAD	8x6	Hard	+	+	+	Cx,Vg H	Atropic	+	5x6.5x4	67.99	T	ME	+	Complex	+	2	5	7	B	TAH BSO	Papillary Serous Cystadenocarcinoma
79	Janaki	55	51053	P	PM	P2L2	NAD	15x18	Cystic	+	-	-	Cx,Vg H	NS	+	10x15x11.5	902.17	S	ME	-	Cystic	-	5	1	6	U	TAH BSO	Benign Cystic Teratoma

80	Tamilarasi	28	15850	P	R	P3L3	NAD	10x12	Cystic	+	-	-	Cx,Vg H	NS	+	9x7.8x5	183.57	S	So	-	Cystic	-	3	0	3	U	ROC	Benign Cystic Teratoma
81	Kalimuthu	60	14925	P	R	Nulli	NAD	10x15	Hard	+	-	+	Cx,Vg H	NS	+	12x10x10	627.6	T	DE	-	Complex	+	5	5	10	U	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
82	Chittammal	35	15438	P	PM	P4L4	NAD	8x9	Variable	+	+	+	Cx,Vg H	NS	+	7x6x8	175.72	S	ME	+	Cystic	+	3	5	8	U	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
83	Nagooramal	35	15876	P	PH	P2L2	NAD	8x8	Cystic	+	-	-	Vault H	.	+	7x7x5.5	140.94	S	So	-	Cystic	-	3	0	3	U	LO	Benign Serous Cystadenoma
84	Ponnupandi	32	17843	P	R	P4L4	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10x6.5x5	169.97	T	So	-	Cystic	-	3	2	5	U	TAH BSO ICO	Benign Mucinous Cystadenoma
85	Subbuthai	43	7373	P	R	P2L2	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x5x6.5	169.97	S	DE	+	Cystic	-	3	3	6	B	TAH BSO ICO	Fibrothecoma
86	Selvarani	25	20743	P	PM	P2L2	NAD	10x12	Cystic	+	-	-	Cx,Vg H	NS	+	10x5x8	209.2	S	So	-	Cystic	-	4	0	4	U	LO	Benign Mucinous Cystadenoma
87	Andiyammal	65	23363	D	PM	P6L5	NAD	20x15	Cystic	+	-	-	Cx,Vg H	Atropic	+	20x15x18	2824.2	S	DE	-	Complex	-	4	4	9	U	TAH BSO ICO	Fibrothecoma
88	Nagamani	30	23343	P	R	Nulli	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x5x8	209.2	S	So	-	Cystic	-	4	0	4	U	RO	Benign Serous Cystadenoma
89	Alagammal	70	22866	P	PM	Nulli	NAD	8x7	Cystic	+	+	-	Cx,Vg H	NS	+	6.8x4x8	113.8	S	So	-	Cystic	-	3	0	3	U	BSO	Benign Serous Cystadenoma
90	Mookammal	55	24426	P,D	R	P4L4	NAD	12x12	Hard	+	+	+	Cx,Vg H	Atropic	+	11.5x9.5x10	571.37	T	ME	+	Complex	+	5	5	10	B	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
91	Nithya	19	3250	P	R	Mulli	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	8.5x10x7.5	333.41	S	So	-	Cystic	-	4	0	4	U	LO	Benign Serous Cystadenoma
92	Rajathi	50	4391	P	R	P2L2	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	9.5x8x6.5	258.36	S	ME	-	Cystic	-	4	1	5	B	TAH BSO	Benign Cystic Teratoma
93	Pandeswari	25	4725	P	R	P2L2	NAD	7x5	Cystic	+	-	-	Cx,Vg H	NS	+	7x5x4.5	82.37	S	So	-	Cystic	-	2	0	2	U	RO	Benign Serous Cyatadenoma
94	Chinnammal	26	2865	P	R	G2P1L1	NAD	10x6	Cystic	+	-	-	Cx,Vg H	NS	+	10x6x8.5	266.73	S	So	-	Cystic	-	4	0	4	U	LSO	Benign Serous Cystadenoma
95	Pandiammal	25	11652	P	R	Primi	NAD	-	-	-	-	-	Cx,Vg H	NS	+	10x6x4.2	131.79	S	So	-	Cystic	-	3	0	3	U	LOC	Benign Serous Cystadenoma
96	Ramalakshmi	45	10640	P,D	R	P3L3	NAD	20x15	Cystic	+	-	-	Cx,Vg H	NS	+	20x21.5x15.2	3418.32	T	ME	-	Complex	-	5	4	9	U	TAH BSO	Benign Mucinous Cystadenoma with borderline malignancy
97	Dhanalakshmi	35	12236	P,D	R	Nulli	NAD	15x13	Cystic	+	-	-	Cx,Vg H	NS	+	12x11x10.5	724.87	S	DE	-	Cystic	-	5	1	6	U	TAH BSO	Benign Endometriotic tumor
98	Bakkiyam	50	16036	D	R	Mulli	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x8.5x9	400.09	S	ME	+	Cystic	-	4	3	7	U	TAH BSO ICO	Benign Serous Cystadenoma
99	Dhanalakshmi	60	14493	P	R	P2L2	NAD	15x15	Cystic	+	-	-	Cx,Vg H	NS	+	14x16.5x12	1449.75	S	So	-	Cystic	-	5	0	5	U	TAH BSO	Benign Mucinous Cystadenoma
100	Pandeswari	22	13801	P,M	R	P6L5	NAD	15x12	Cystic	+	-	-	Cx,Vg H	NS	+	13.5x12x10	847.2	S	So	-	Cystic	-	5	0	5	U	RO	Benign Serous Cystadenoma

101	Seethalakshmi	49	14778	P,D	R	P2L1	NAD	12x12	Cystic	+	-	-	Cx,Vg H	NS	+	12x11.5x9	649.56	T	DE	-	Cystic	-	5	1	6	U	TAH BSO ICO	Benign Papillary Seromucinous Cystadenoma
102	Lakshmi	40	18652	P,D	R	P2L1	NAD	10x13	Cystic	+	-	-	Cx,Vg H	NS	+	13x10.8x9	660.86	S	So	-	Cystic	-	5	0	5	U	TAH BSO ICO	Benign Mucinous Cystadenoma
103	Chitra	23	22712	P	R	P1L1	NAD	10x8	Cystic	+	-	-	Cx,Vg H	NS	+	10.8x7.4x8	334.38	S	So	-	Cystic	-	4	0	4	U	LOC	Benign Mucinous Cystadenoma
104	Muthulakshmi	16	23184	P	R	Nulli	NAD	8x6	Cystic	+	-	-	Cx,Vg H	NS	+	4x7x6.2	90.79	S	So	-	Cystic	-	2	0	2	U	LOC	Benign Serous Cystadenoma
105	Eswari	25	2813	,M, MDp	R	P3L2	NAD	7x8	Cystic	+	-	-	Cystocele	NS	+	7x3.2x4	46.86	S	So	-	Cystic	-	1	0	1	U	RO	Benign Serous Cystadenoma
106	Meenambal	62	5906	P	R	P4L5	NAD	25x20	Variable	+	+	-	Cx,Vg H	NS	+	20x18x19.5	3671.46	T	ME	+	Complex	-	5	4	9	B	Inoperable	Adenocarcinoma
107	Guruvu	85	5931	P	R	P4L4	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	11.2x10x9.4	550.61	T	DE	-	Complex	-	5	4	9	U	TAH BSO	Poorly Differentiated Papillary Carcinoma
108	Sudha	39	10703	P,M	R	P2L2	NAD	18x16	Cystic	+	-	-	Cx,Vg H	NS	+	16x18x16.5	2485.3	S	So	-	Cystic	-	5	0	5	U	TAH BSO	Benign Serous Cystadenoma
109	Muthupandiammal	22	10866	D,M	R	P2L2	NAD	13x11	Cystic	+	-	-	Cx,Vg H	NS	+	11x6x7	462	S	DE	-	Cystic	-	4	1	5	U	TAH BSO ICO	Benign Serous Cystadenoma
110	Kamalam	52	12860	P,D	R	P5L3	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	10x9x6	282.42	S	So	-	Cystic	-	4	0	4	U	TAH BSO	Benign Serous Cystadenoma
111	Kalaiammal	41	12905	P	PH	P3L3	NAD	10x6	Cystic	+	-	-	Vault H	.	+	8x6x5	125.52	S	So	-	Cystic	-	3	0	3	U	TAH BSO	Benign Serous Cystadenoma
112	Lakshmi	32	59852	P, V	R	P2L2	NAD	10x12	Cystic	+	-	-	Cx,Vg H	NS	+	7.6x5x4	79.49	S	So	-	Cystic	-	2	0	2	U	TAH BSO	Benign Serous Cyatadenoma
113	Jothi	20	18229	P,M	R	Primi	NAD	12x7	Cystic	+	-	-	Cx,Vg H	16 wks	+	10x8x6.5	271.96	S	ME	-	Cystic	-	4	1	5	U	RO	Benign Cystic Teratoma
114	Vairakkal	56	17892	P,MA	IR	P4L4A1	NAD	25x15	Cystic	+	-	-	Cx,Vg H	NS	+	20x18.5x21.2	4102.41	S	DE	-	Cystic	-	5	1	6	U	TAH BSO ICO	Mucinous Papillary Cystadenocarcinoma
115	Ambigavathy	30	61872	D, C, PE	IR	P2L2	NAD	20x15	Cystic	+	+	+	Cx,Vg H	NS	+	15.5x12x10	972.78	T	ME	+	Complex	+	5	5	10	U	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
116	Murugayee	60	19139	P, M, LA	R	P4L4	NAD	20x18	Cystic	+	+	-	Cx,Vg H	NS	+	15x16.5x18	2329.98	T	ME	+	Complex	+	5	5	10	U	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
117	Laxiammal	60	19116	D	R	P3L2	NAD	30x20	Cystic	+	+	-	Cx,Vg H	NS	+	20x18x16	3012.48	T	So	-	Complex	+	5	5	10	U	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
118	Mariammal	52	6309	MA	IR	P3L3	NAD	6x8	Cystic	+	-	-	Cx,Vg H	Bulky	+	6x6x5	94.14	T	DE	-	Cystic	+	2	5	7	B	TAH BSO ICO	Papillary serous tumor of borderline malignancy
119	panju	42	8617	P	R	P5L5	NAD	10x10	Hard	+	-	-	Cx,Vg H	NS	+	10x11x9	517.77	T	DE	+	Complex	+	5	5	10	B	TAH BSO ICO	Mucinous cystadenocarcinoma
120	Dhanalakshmi	32	927	P,D	R	P2L2	NAD	20x15	Cystic	+	-	-	Cx,Vg H	NS	+	19.6x21x18	3874.8	S	So	-	Cystic	-	5	0	5	U	RO	Benign Mucinous Cystadenoma
121	Lakshmi	35	2076	P	R	P5L1	NAD	10x10	Cystic	+	-	-	Cx,Vg H	NS	+	12x10x11	690.36	S	So	+	Cystic	-	5	2	7	U	TAH BSO	Benign Cystic Teratoma

122	Meera	27	5782	P	R	P2L2	NAD	6x7	Cystic	+	-	-	Cx,Vg H	NS	+	5.2x5x4	54.39	S	So	-	Cystic	-	2	0	2	U	RO LSO	Benign Serous Cystadenoma
123	Priya	23	9028	P	R	Nulli	NAD	9x9	Cystic	+	-	-	Cx,Vg H	NS	+	9x8.2x7	270.18	S	So	+	Cystic	-	4	2	6	U	RO	Benign Cystic Teratoma
124	Bakkiyam	30	9542	P	R	P1L1	NAD	7x7	Cystic	+	-	-	Cx,Vg H	NS	+	7.2x5x6	112.96	S	So	-	Cystic	-	3	0	3	U	LO	Benign Serous Cystadenoma
125	Guruvammal	45	9033	P,D	R	P2L2	NAD	20x25	Cystic	+	-	-	Cx,Vg H	NS	+	21x18x19.5	3855.03	S	ME	-	Cystic	-	5	1	6	U	TAH BSO ICO	Benign Mucinous Cystadenoma
126	Paapa	47	12433	P	R	P4L4	NAD	6x8	Cystic	+	-	-	Cx,Vg H	NS	+	4x6x8	100.42	S	So	-	Cystic	-	2	0	2	U	LOC	Benign Serous Cystadenoma
127	Sundareshwari	35	15786	P	R	P1L1	NAD	9x10	Cystic	+	-	-	Cx,Vg H	NS	+	9x8x6	225.93	S	So	-	Cystic	-	4	0	4	U	RO	Benign Serous Cystadenoma
128	Selvi	28	9013	P	R	P3L3	NAD	20x20	Cystic	+	-	-	Cx,Vg H	NS	+	20x21.5x16	3598.24	S	DE	-	Cystic	-	5	1	6	B	TAH BSO ICO	Ovarian Fibrothecoma
129	Chitra	25	9109	P	R	P2L2	NAD	20x15	Cystic	+	-	-	Cx,Vg H	NS	+	20x22.5x18	4257.22	S	ME	-	Cystic	-	5	1	6	U	LSO	Benign Mucinous Cystadenoma
130	Lakshmi	55	8001	P	R	P2L0	NAD	8x6	Cystic	+	-	-	Cx,Vg H	NS	+	8x7x6.6	193.3	S	So	-	Cystic	-	3	5	8	B	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
131	Mariammal	52	6309	MA	IR	P3L3	NAD	6x8	Cystic	+	-	-	Cx,Vg H	Bulky	+	6x6x5	94.14	S	So	-	Cystic	-	2	5	7	U	TAH BSO ICO	Papillary serous tumor of borderline malignancy
132	Selvam	45	12834	P	R	P2L1	NAD	8x8	Cystic	+	-	-	Cx,Vg H	NS	+	8x7x6	175.7	S	So	-	Cystic	-	3	5	8	B	TAH BSO ICO	Papillary Serous Cystadenocarcinoma
133	Vellathai	48	4744	P,D	R	P4L2A2	Cachetic	20x15	Variable	-	+	+	Cx,Vg H	NS	+	20x12x8.5	1066.92	T	DE	+	Complex	+	5	5	10	B	TAH BSO ICO	Adenocarcinoma
134	Karupayee	60	65607	P	PM	P4L4	NAD	12x12	Hard	-	-	+	Cx,Vg H	Atropic	+	12x14x8.5	746.84	T	ME	-	Cystic	+	5	5	10	B	TAH BSO ICO	Mucinous Cystadenocarcinoma
135	Ramalan	68	8234	P	PM	P4L4	NAD	10x15	Firm	-	-	-	Cx,Vg H	Atropic	+	13x8x10.5	571.11	T	So	+	Cystic	-	5	3	8	B	TAH BSO ICO	Brenner tumor
136	Sathya	13	8078	D	R	Nulli	NAD	20x15	Hard	-	-	+	Cx,Vg H	NS	+	18x15x12.5	1765.13	T	ME	+	Complex	+	5	5	10	U	RSO ICO	Mucinous Cystadenocarcinoma